APR 26 1929

Vol. IV APRIL, 1929 No. 4

THE AMERICAN HEART JOURNAL

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PUBLISHED BI-MONTHLY

UNDER THE EDITORIAL DIRECTION OF

THE AMERICAN HEART ASSOCIATION

LEWIS A. CONNER - - - - - - Editor
HUGH McCULLOCH - - Associate Editor

PUBLISHED BY THE C. V. MOSBY COMPANY, 3523-25 PINE BLVD., ST. LOUIS, U. S. A.

Entered at the Post Office at St. Louis, Mo., as Second Class Matter.

The American Heart Journal

CONTENTS FOR APRIL, 1929

Original Communications

Cardiac Pain. A Consideration of Its Nosology and Clinical Associations. By Robert L. Levy, M.D., New York City, N. Y. ---------- 377

Pain Due to Temporary Occlusion of the Coronary Arteries in Dogs. By J. Frank Pearcy, M.D., New York City, Walter S. Priest, M.D., and C. M. Van Allen, M.D., Chicago, Ill. ---------- 390

The Relation of Clinical, Including Electrocardiographic, Phenomena to Occlusion of the Coronary Arteries Based on the Observation of a Case. By Harold J. Stewart, M.D., New York City, N. Y. ---------- 393

The Action of Digitalis in Complete Heart-Block. Its Toxic Influence on the Idioventricular Rate and Rhythm. By Sidney P. Schwartz, M.D., New York City, N. Y. ---------- 406

The Incidence and Significance of Electrocardiograms Showing the Features of Left Axis Deviation and QRS of Normal Duration With Inverted T_1 and Upright T_3. By Drew Luten, M.D., and Edward Grove, M.D., St. Louis, Mo. ---------- 431

The Pre-Employment Examination of the Cardiac Worker. By C. H. Watson, M.D., New York City, N. Y. ---------- 442

Mortality and Morbidity From Heart Disease Under Industrial and Group Insurance. By Wade Wright, M.D., New York City, N. Y. ---------- 448

Statistical Studies Bearing on Problems in the Classification of Heart Diseases. V. Heart Disease Among Ex-Service Men. By Philip B. Matz, M.D., Washington, D. C. ---------- 455

The Elimination From the Electrocardiogram of the Effects of Alternating Current of Power and Lighting Lines. By Russell A. Waud, M.D., Ph.D., London, Ontario ---------- 477

Department of Clinical Reports

Pleural Effusion Associated With Congestive Heart Failure Localized in an Interlobar Space. By Edgar F. Kiser, M.D., Indianapolis, Indiana ---- 481

Retroperitoneal Abscess of Splenic Origin in a Case of Subacute Bacterial Endocarditis. By Edward Weiss, M.D., Philadelphia, Pa. ---------- 484

Department of Reviews and Abstracts

Selected Abstracts ---------- 486

The American Heart Journal

VOL. IV APRIL, 1929 NO. 4

Original Communications

CARDIAC PAIN

A CONSIDERATION OF ITS NOSOLOGY AND CLINICAL ASSOCIATIONS*

ROBERT L. LEVY, M.D.
NEW YORK, N. Y.

PAIN in the region of the heart is a common complaint. Of the last five hundred patients who have come to the consulting room, 15 per cent have described pain referred to the sternum or precordium either as the chief source of discomfort or as one of several complaints. Its significance for the patient may be trivial or grave; but since Heberden's memorable communication, the occurrence of such pain has come to carry with it, in the minds of the laity and indeed, in the judgment of many physicians as well, the sinister suggestion of sudden death.

HISTORICAL

It was on July 21, 1768, that William Heberden, a distinguished London practitioner, read before the Royal College of Physicians a paper entitled *Some Account of a Disorder of the Breast.*[1] Because our knowledge of this condition and also the still unfinished controversies concerning its nosology were initiated by his masterly description, it is desirable to give quotations from the original text.

"There is a disorder of the breast, marked with strong and peculiar symptoms, considerable for the kind of danger belonging to it, and not extremely rare, of which I do not recollect any mention among medical authors. The seat of it, and sense of strangling and anxiety with which it is attended, may make it not improperly be called Angina Pectoris.

"Those, who are afflicted with it, are seized, while they are walking, and more particularly when they walk soon after eating, with a painful and most disagreeable sensation in the breast, which seems as if it would take their life away, if it were to increase or to continue: the moment they stand still, all this uneasiness vanishes. In all other respects, the patients are, at the beginning of this disorder, perfectly well, and in particular have no shortness of breath, from which it is totally different. . . . .

*From the Department of Medicine, College of Physicians and Surgeons of Columbia University and the Presbyterian Hospital.

Read before the Rochester Medical Association and Rochester Academy of Medicine, Rochester, N. Y. November 7, 1928.

"When I first took notice of this distemper, and could find no satisfaction from books, I consulted an able physician of long experience, who told me that he had known several ill of it, and that all of them had died suddenly. This observation I have reason to think is generally true of such patients, having known six of those, for whom I had been consulted, die in this manner. . . . But though the natural tendency of this illness be to kill the patients suddenly, yet unless it have a power of preserving a person from all other ails, it will easily be believed, that some of those, who are afflicted with it, may die in a different manner, since this disorder will last, as I have known it more than once, near twenty years, and most usually attacks only those who are above fifty years of age. . . . .

"The os sterni is usually pointed to as the seat of this malady, but it seems sometimes as if it was under the lower part of it, and at other times under the middle or upper part, but always inclining more to the left side, and sometimes there is joined with it a pain about the middle of the left arm. What the particular mischief is, which is referred to these different parts of the sternum, it is not easy to guess, and I have had no opportunity of knowing with certainty."

It was the astute Edward Jenner who first threw light on the nature of this "mischief." In 1776, John Hunter had his second heart attack, and was visited at Bath by his pupil, Jenner. To Heberden, Jenner then wrote a letter, giving his diagnosis of Hunter's malady and suggesting, for the first time, the probable association of coronary artery disease and cardiac pain.[2] In a later letter, published by Parry in 1799,[3] Jenner gives a fuller account of his findings and states that he did not publish his views because he feared that they might be a source of worry to his friend, Hunter. This pathological description is also worth quoting.

"The first case I ever saw of Angina Pectoris, was that in the year 1772, published by Dr. Heberden with Mr. Hunter's dissection. There, I can almost positively say, the coronary arteries were not examined. Another case of a Mr. Carter, at Dursley, fell under my care. In that, after having examined the more important parts of the heart, without finding anything by means of which I could account either for his sudden death, or the symptoms preceding it, I was making a transverse section of the heart pretty near its base, when my knife struck against something so hard and gritty, as to notch it. I well remember looking up to the ceiling, which was old and crumbling, conceiving that some plaister had fallen down. But on a further scrutiny the real cause appeared: the coronaries were become bony canals. Then I began a little to suspect. Soon afterwards, Mr. Paytherus met with a case. Previously to our examination of the body, I offered him a wager that we should find the coronary arteries ossified. This, however, proved to be not exactly true; but the coats of the arteries were hard, and a sort of cartilaginous canal was formed within the cavity of each artery, and there attached, so however as to be separable as easily as the finger from a tight glove. At this very time, my friend Mr. John Hunter began to have the symptoms of Angina Pectoris too strongly marked upon him; and this circumstance prevented any publication of my ideas on the subject, as it must have brought on an unpleasant conference between Mr. Hunter and me. I mentioned both to Mr. Cline and Mr. Home, my notions of the matter, ... but they did not seem to think much of them. When, however, Mr. Hunter died, Mr. Home very candidly wrote to me, immediately after the dissection, to tell me I was right."

Whether priority for the description of what Heberden termed "Angina Pectoris," should be given to Seneca (1605), Morgagni (1761) or to the Frenchman, Rougnon (Feb. 1768) is a matter of some historic interest but is not pertinent at this time. How the clinical concept which Heberden portrayed was viewed by those who followed, and in what manner controversial discussion arose, is directly related to the development of present day thought. Only a few of the steps in the argument will be touched upon.

Parry, in 1799,[3] was among the first to sense the difficulty of adhering closely to the criteria laid down by Heberden, and to point out that cases described by other observers as angina pectoris did not conform to one pattern. He believed that the symptoms in one reported case were due to hydropericardium, in another to aneurysm of the aorta and in a third to "suppuration between the two bags of the pleura, where they constitute the mediastinum." In other instances, he questioned the accuracy of the diagnosis, saying that the "cases of MacBride and Smith of Dublin, are evidently cases of palpitation of the heart, such as every physician of extensive practice must have often seen"; another, "was a mere affection of the respiratory organs, a violent spasmodic asthma, . . . which never occurs in the pure Angina Pectoris." Parry then proceeded to add to the confusion by concluding that "the Angina Pectoris is in reality a case of fainting. . . . All the circumstances in the Angina Pectoris preceding the actual Syncope are approaches towards it; and in every uncombined and recent case, like those which I have described, the patient probably dies with no other symptoms than those which shew an irrecoverable diminution of the motion of the heart." Parry's monograph is entitled *An Inquiry into the Symptoms and Causes of the Syncope Anginosa, commonly called Angina Pectoris.* This is the first sign of dissension from Heberden's terminology.

Hope[4] in 1839, heads a chapter in his book *Neuralgia of the Heart, or Angina Pectoris,* in "presenting the train of symptoms which have been denominated by Dr. Heberden *angina pectoris.*" He points out that "different physicians have found it connected with different organic lesions or states, and each has supposed it to be occasioned by that, with which he has most frequently found it co-exist. Dr. Parry, and after him Burns and Kreysig, ascribe it to ossification of the coronary arteries; Dr. Hooper, to affections of the pericardium; Dr. Hosack to plethora; Dr. Darwin, to asthmatic cramp of the diaphragm; Drs. Butler, Macqueen, and many others, have regarded it as a particular species of gout; Dr. Latham has found it connected with enlargements of the abdominal viscera, while the thoracic viscera were sound; and Heberden, having found it both connected and unconnected with organic disease, thinks that its cause has not been traced out, but that it does not seem to originate necessarily in any structural derangement

of the organ affected." Hope expressed the opinion that "it may originate in any cause, whether organic or functional, capable of *irritating* the heart, or of rendering it morbidly susceptible of irritation; and as structural disease of the organ has this effect more than other cause, it is that on which the malady, in its severer forms, is most frequently dependent."

Latham,[5] 1845, flatly refused to accept angina in the sense of Heberden. Says he: "There is one eminent instance in which an assemblage of symptoms is thus made to bear the name of a disease; angina pectoris." There follows a description of the variety of pathological states found at necropsy in cases so diagnosticated during life; as well as the statement that "it has existed where no form of disease or disorganization whatever has been found either in the heart or in the blood vessels nearest to it."

In the experience of the English observers, angina was a rare affection. In contradistinction to the rarity of genuine angina pectoris, Walshe[6] in his textbook, says that he frequently met "with a form of complaint combining in a minor degree many of the characters of angina; and to this imitation of the true disease I propose to give the name of pseudo-angina." Walshe found pseudo-angina as an attendant on various diseases: gout, rheumatism, anemia, hysteria, spinal irritation, epilepsy, intercostal and mammary neuralgia. The points of differential diagnosis between genuine and pseudo-angina are but briefly mentioned. Yet this author is apparently the first to express the idea that cardiac pain may occur in many conditions in which the heart is not primarily the seat of the trouble.

Like Hope, Laennec[7] classifies angina pectoris as a neuralgia of the heart. Though familiar with the necrospy findings of the English authors and having found various diseased states of the heart and aorta in patients with this complaint, he was particularly impressed by those cases showing no demonstrable lesions on post-mortem examination. He was inclined to ascribe the painful sensations to an affection of the cardiac nerves, vagus or sympathetic, analogous to neuralgias elsewhere in the body. Other nerves, cervical and thoracic, he believed, might likewise be involved, either "sympathetically" or through anastomatic communications with those primarily concerned.

In his monograph on *Angina Pectoris and Allied States*, Osler[8] reiterates Latham's sentiments in saying: "Angina pectoris is not a disease, but a syndrome or symptom group (without etiological or anatomical foundation) associated with complex conditions, organic or functional, of the heart and aorta. Pain about the heart of an agonizing character, occurring in paroxysms, is the dominant feature of all varieties of the syndrome. Used to define paroxysmal attacks of pain in the chest—breast-pang—we employ the term generically, qualifying the varieties by such names as true, false, hysterical and vasomotor."

But such generic use of the term has not served to clarify the picture. The distinctions between syndrome and disease are not apparent in this definition.

Among those who gave much scholarly thought to this subject was the late Sir T. Clifford Allbutt, as evidenced by his two volumes on *Diseases of the Arteries, including Angina Pectoris.* Several years ago, having published a short paper on cardiac pain,[9] I ventured to write to Cambridge to Sir Clifford asking for his comment. On October 7, 1924, not long before his death, he answered in a letter, expressing what were perhaps his last ideas on this subject. I am taking this opportunity to make them public.

"The difficulty of defining angina pectoris is great because it has so many simulations, and it is hard to exclude these; e.g., tobacco angina, neurotic, and (I think) a tiresome wedge of wind in the lower esophagus which oppresses certain persons painfully on walking—though they can climb hills and stairs, etc., without any—or any more distress and it does not *stop* the patient; he *can* go on, although oppressed. This is commoner in women? I was far from opposing your point of view, one I have urged myself in many contexts. I would have such words as 'asthma' kept strictly to their own (or one) meaning (not 'cardiac' asthma, etc.). So I think *angina pectoris* should be pinned down to substernal (or epigastric) pain, (very rarely in the 'precordial' area) apt to shoot in certain definite directions, and having nearly always a sinister touch about it. It *stops* a man to vague apprehension. This of course is no 'definition'; but what disease *can you define?* It can only be a *description;* longer or shorter. You cannot *define* even a cat. And please do not put 'morbid entity' upon me; it is one of the last remnants of medical ontology—a relic of terminology. But an angina pectoris due to syphilitic aortitis *only* there is, and frequently. Comparatively young persons *don't die* of angina—indeed *directly no one* does: he dies of heart shock—vagus- or ventricular fibrillation due to the afferent pain. The young, sound heart does not *die of it,* horribly as the patient may suffer. We all agree about the spinal segments concerned and referred pains from the pathologic seat of the pain. Not Mackenzie only but also Head and others have worked these out long ago. Mackenzie contends the pain *originates* in the cardiac muscle; but no *afferent nerve* enters muscle—the heart when touched or pricked is insensitive. The pain arises in the *investments* which are *full of* sensory end-organs—the pain is due to a drag upon these—pericardial rarely, aortic in the large majority. Pain in skeletal muscles arises in the investments and fibrous scaffold—the heart muscle has no such fibrous strands. You will see in my book I do not attribute *all* cases of angina pectoris to the periaortic tissue—a few I describe as pericardial. Coronary artery disease is concerned only with *death* in angina pectoris; not with the seat of its origin. 'Hypertension' does not cause angina pectoris if the aorta is sound; it sometimes does arouse it in an aorta not quite sound, but which would not have resented moderate pressure. Aneurysm is merely a cause of aortic disease. Thrombosis may cause exquisite pain if sudden—e.g. embolic, because it puts the arterial investment to sudden strain. In a word, angina pectoris is *not* 'cardiac' pain except in so far as you include in some few cases its outer coat. Neuroses *are not* angina pectoris at all; they are generally intercostal neuralgias. The common submammary ache of cardiacs and menorrhagias, etc., is not angina pectoris. I agree the *clinical* distinctions ought not often to deceive—but a few are hard to read on *one single consultation.* Fasten angina pectoris down to the real thing and you get as much 'definition' as in biology we ever can."

On a small slip of paper, enclosed in the envelope with the body of the letter, was the following: "Angina pectoris is a pain, sometimes slight, sometimes agonizing, arising usually under the sternum and referred in certain definite directions, due to stretching of the outer coat of a morbid aorta; or in a few cases of that of the heart itself. This is my first hasty suggestion for your 'definition.'"

THE MECHANISM OF HEART PAIN

How a painful sensation is produced in the heart is not clear. The myocardium is insensitive to stimuli which are ordinarily productive of discomfort in the periphery of the body. Afferent nerve endings have been found in the outer coat of the blood vessels and in the pericardium. Recent ingenious experiments on dogs[10] have demonstrated that compression of the coronary vessels and immediately adjacent tissue always causes evidences of pain and sometimes produces salivation, vomiting, and changes in the electrocardiogram.

Some sixty-three hypotheses concerning the cause of the symptoms in angina pectoris were collected and tabulated by Huchard.[11] Three theories of pain production in the heart seem worthy of consideration. Allbutt[12] regarded pain in the region of the heart as due to a lesion in the supracardiac aorta, which he termed the "anginiferous area." The lesion, in his opinion, causes "angina" when it penetrates to the outer fibrous investment of the vessel, tension being the factor causing irritation of the nerve terminals. By Mackenzie,[13] the importance of exhaustion of the myocardium was stressed. His hypothesis was summed up in stating that, "while we can reason that pain is the result of exhaustion when the blood supply to the muscle is deficient, we can also conclude that pain results when exhaustion is produced from any other cause, such as great exertion by a healthy heart, or by relatively slight exertion where the muscle is diseased." It was Allan Burns,[14] as Osler has pointed out, who, over a century ago, ascribed the symptoms to anemia of the heart muscle. "Such a state of the arteries of the heart," says he, referring to atheroma, "must impair the function of that organ." In a careful review of the evidence, Keefer and Resnik[15] recently have reached the same conclusion: "the angina pectoris of Heberden has but one cause, anoxemia of the myocardium." This point of view, though at present lacking proof, suggests cogent arguments in its favor. It does not explain all cases. Perhaps more than one mechanism may be concerned in varying circumstances.

THE CLINICAL ASSOCIATIONS OF CARDIAC PAIN

It is evident from the fragmentary account which has been given of the development of thought concerning Heberden's angina, that confusion exists concerning its precise definition. Is every case of paroxysmal breast pain to be so diagnosticated? If not, what are the cri-

teria for this condition? If one of these criteria be sudden death, is it only after the patient's demise that the diagnosis may be made with certainty? For many years, a variable pathological basis has been recognized. The discussion as to the distinctions between "symptom group" or syndrome, and disease has been carried on to no practical purpose. A descriptive term meaning literally, "strangling in the chest," has been perpetuated in spite of protest and acknowledged dissatisfaction. A serious nosologic consideration of the matter is not merely "factitious hair splitting," as Sir Clifford Allbutt would have us believe. Looseness in classification makes for careless thinking; and careless thinking leads to inaccuracies in all forms of scientific procedure.[16]

The pain may vary in its severity, its distribution, and in its relation to exertion. The degree of effort necessary to induce it depends, in a measure, on the individual's sensitiveness to unpleasant stimuli. It is customary to describe as part of the clinical picture of angina a sense of impending dissolution experienced by the patient—the *angor animi* of the older writers. Whether or not this feeling of the nearness of death is present depends largely on the nervous constitution of the sufferer. The work of Gross,[17] and of Oberhelman and Le Count[18] has demonstrated that the coronary arteries, in different subjects, show considerable variation in their origin, distribution, and anastomoses. It follows that the heart as a whole must present functional differences corresponding to these variations when the coronary vessels are diseased—an observation which may account, in part, for the multiplicity of clinical pictures under these circumstances. The rôle of the Thebesian veins in contributing toward an adequate blood supply for the heart muscle must also be taken into account.[19] In the event of gradual closure of the orifices of the coronary arteries, the Thebesian vessels, if allowed time to adapt themselves, can supply the myocardium with sufficient blood to enable it to maintain an efficient circulation. Many factors operate in the determination of the symptoms and signs in a given case.

Those desirous of retaining the term angina, stoutly maintain that it denotes a sharply defined clinical picture, distinguishable from its imitators. So, in contradistinction to true, primary, or major angina, they have described false or pseudo-angina, secondary angina, minor angina, the mock anginas, hysterical angina, angina vasomotoria, tobacco angina and finally, angina sine dolore. Truly, an imposing array of impostors! Differential diagnosis in many medical conditions may be difficult; yet we do not speak of "false appendicitis" because other disturbances in the abdomen may simulate inflammation of the appendix. Our efforts are directed toward describing and correlating symptoms, signs, and anatomical states in order to become familiar with a

train of events which we then call a *disease*. In this concept of disease is implied disorder of both function and structure.

In view of these considerations, and until further knowledge may make a more precise terminology feasible, it is suggested that the use of the term "angina pectoris" be discontinued. In its place, "cardiac pain" may be employed, in each instance qualified by notation of the structural or functional changes with which such pain is found to be associated. A classification formulated on this basis is not, strictly speaking, etiological. But it is workable and encourages more precise diagnosis. Inevitably, better therapy and more accurate prognosis must follow. Such a classification is the following:

CLINICAL CONDITIONS IN WHICH CARDIAC PAIN MAY BE ENCOUNTERED

I. Affections of the Coronary Arteries.
   1. Stenosis.
      a. Arteriosclerotic.
      b. Syphilitic.
   2. Occlusion.
      a. Arteriosclerotic.
      b. Syphilitic.
      c. Thrombotic.
      d. Embolic (rare).

II. Affections of the Aorta.
   1. Aortitis (with or without Dilatation).
      a. Syphilitic.
      b. Rheumatic.
   2. Arteriosclerosis (with or without Dilatation).
   3. Aneurysm.
      a. Syphilitic.
      b. Arteriosclerotic.
   4. Rupture.

III. Cardiac Valvular Disease.
   1. Aortic Insufficiency.
   2. Mitral Stenosis.
   3. Congenital Heart Disease.

IV. Pericarditis.
   1. Serofibrinous.
   2. Serous.
   3. Adhesive.

V. Poisoning of the Heart by:
   1. Tobacco.
   2. Coffee.
   3. Tea (uncommon)

VI. Disorders of the Hematopoietic System.
   1. Anemia (Primary or Secondary).
   2. Erythremia.

VII. Endocrine Disorders.
1. Thyrotoxicosis.
2. Myxedema.
3. Addison's Disease.

VIII. Cardiac Neurosis (Effort Syndrome).

IX. Pathogenesis Undetermined.

The history and necropsy findings in one unusual case are here given:

A man, aged forty-three years, an electrician, entered the Presbyterian Hospital on December 25, 1924, for the ninth time. In 1918 and again in 1922, he was treated in the ward for influenza. He never had any symptoms of heart disease. His only previous illnesses were typhoid at the age of fourteen and pneumonia at the age of twenty-six. There was no history of syphilis and the Wassermann reaction was negative. In January, 1924, he was admitted with a diagnosis of lobar pneumonia involving the left lower lobe. This was confirmed by x-ray examination. On the second day in hospital, he was suddenly seized with excruciating pain in the precordial area, radiating to both arms. His face was ashen, his lips cyanotic, but there was no dyspnea. The heart sounds became inaudible for a few moments; the blood pressure, twenty-five minutes after the attack, was 70 mm. systolic; 50 mm. diastolic. Morphine and cardiac stimulation brought about comfort and recovery. That evening, a similar, though less severe, attack occurred, relieved by amyl nitrite. An electrocardiogram showed no deviation from the normal. The leucocytes rose from 15,000 to 22,000. A diagnosis of coronary thrombosis was made, although previous examination of the heart and arteries was quite negative. He complained of mild substernal pain on two subsequent occasions and went home on the twelfth day after admission.

Following these episodes the history during the next eleven months was one of progressive cardiac disability. Nocturnal dyspnea appeared and was accompanied by a sense of substernal oppression. Severe attacks of precordial pain were induced on slight effort, making work, even of a light nature, impossible. On six occasions, because of the paroxysms of pain, he was brought to the hospital, sometimes in an ambulance. Many electrocardiograms were taken, some of them showing changes in the T-waves in Leads I and II which were interpreted as indicating active disturbances in the myocardium. Teleroentgenograms showed no cardiac enlargement. There were no signs of valvular disease. As the number of observations increased, the opinion was expressed that coronary disease, with narrowing of the lumen of the vessels, was the pathological lesion responsible for the symptoms. The blood pressure was usually about 124/80 between attacks. Blood counts were normal.

The man was ambitious and eager to work for his wife and six children. The attacks of pain were described as "crushing," "pressing" and "vise-like," precordial or substernal and usually radiating down both arms. Nitrites always afforded immediate relief. At times, nausea and vomiting followed a paroxysm. Cervical sympathectomy was advised with the hope that he might be returned to economic usefulness.

The operation was undertaken on December 26, 1924, by Dr. W. G. Penfield, about eleven months after the first painful seizure. Ether was administered by the intranasal method. There was some handling of the vagus, but no change in pulse rate was noted at these times. The middle and inferior sympathetic ganglia on the left side were exposed and their branches were cut or torn out. The superior cardiac nerve was cut and the left sympathetic chain was severed just below the superior cervical ganglion, the ganglion being left in place. The wound was closed.

An incision was made on the right side preparatory to operating on the right sympathetic chain. Suddenly, the heart rate increased and the pulse became weak. Respirations came at longer intervals and were gasping. Adrenalin was injected into the heart, but in spite of artificial respiration with a pulmotor, circulation and respiration ceased abruptly. In the opinion of the operator, death appeared to be due to sudden cardiac failure.

Necropsy was performed by Dr. Paige, two and a half hours after death, and was limited to the thorax. The heart weighed 340 grams. Except for a few small sclerotic plaques in both coronary arteries, often seen in persons of this age, the findings were negative. There was no narrowing of the lumen of the vessels. In the arch of the aorta were similar tiny yellow plaques, the largest only 1 or 2 mm. in diameter. Microscopic examination of the tissues was made by Dr. A. M. Pappenheimer. The heart muscle was normal. At one place, the intima of a section of the coronary artery was very slightly thickened. The intima of the aorta showed a small area of necrosis. The media, in its outer third, contained one small area of scarring with a few infiltrating leucocytes. There was no rupture of the elastic fibers. Microscopic examination of the sympathetic ganglia removed at operation was negative. Dr. Pappenheimer's final note states that, "the autopsy discloses no anatomical basis for the anginal attacks."

PROGNOSIS

In no group of cases is the estimation of life expectancy more difficult. The patients with coronary disease may die in their first attack, as did Thomas Arnold, the schoolmaster of Rugby, or like John Hunter, may survive for twenty years, "his life in the hands of any rascal who chose to annoy or tease him." Experience has stressed two points: first, extensive sclerosis of the coronary vessels may exist without cardiac enlargement or significant changes in the electrocardiogram; second, it is precisely in those patients in whom relatively little is to be made out in the heart on physical examination, that early, sudden, and unexpected death not infrequently occurs. This fact was well exemplified in the case of a man, sixty-five years of age, with paroxysms of pain on slight effort, localized to the midsternal region, of three months' duration. There was moderate retinal and peripheral sclerosis. The blood pressure was 126 mm. Hg. systolic; 74 diastolic. The heart was not enlarged and there was a short systolic whiff at the apex. An electrocardiogram showed inversion of the T-waves in Leads I and II. The clinical diagnosis was sclerosis of the coronary arteries. During the year following his first visit, he improved markedly, and was able to walk four or five blocks with only an occasional precordial twinge. He took an ocean voyage to the Barbadoes and on the steamer was entirely free from pain. On his return he went up the subway stairs without discomfort. The T-wave in Lead II became upright in the graphic records. Three weeks after an office examination, I was called hurriedly to the Pennsylvania Railroad Terminal. He was going on a holiday to Atlantic City with his wife and while standing at the ticket window, purchasing his accommodations, fell over dead. Necropsy was not performed.

On the other hand, there is a group of patients with enlarged heart and electrocardiographic signs of serious myocardial damage, who survive their cardiac disability to die of another cause. For example, a man sixty years of age likewise suffered from attacks of pain induced by effort. The heart was markedly enlarged; there was no hypertension. The retinal and peripheral arteries were distinctly sclerosed. A large series of electrocardiograms showed persistent intraventricular block, with varying T-wave changes and occasional ventricular premature beats. He took several trips to Europe and continued his business as a stock broker, though never free from discomfort, taking nitroglycerin several times each day. Six years after the onset of his symptoms, he had a cerebral hemorrhage in bed during the night and died within twenty-four hours without regaining consciousness.

TREATMENT

If pain be regarded as an expression of a disturbed functional or structural state, therapy must be directed toward correcting or alleviating the basic disorder. When possible, this is the plan generally followed in medical and surgical practice; it is the logical procedure when the pain originates in the heart. A detailed account of the management of patients suffering from the various conditions which give rise to cardiac pain is not within the scope of this essay. The attempt to induce symptomatic relief through surgery of the cervical sympathetic chain merits, however, a word of comment.

Operative treatment of cardiac pain was proposed by François Franck in 1899 and first carried out by Jonnesco in 1916. Cutler[20] has recently summarized the experience up to 1927. At the Presbyterian Hospital, only five patients have had cervical sympathectomy, and reports of a sufficient number of accurately observed cases are not available in the literature to justify dogmatic conclusions. Thus far, the results have been variable and, on the whole, disappointing. One reason for lack of uniformity has been the fact that different observers have operated upon patients with pain associated with many different clinical conditions. It is generally conceded that patients with coronary disease are poor subjects for surgery, and it is in this group that the worst sufferers are found. Many of the cases reported as cured after sympathectomy have been followed for only a short time; others undoubtedly would have improved under medical treatment, provided an accurate diagnosis had been made. Until more is known concerning the mode of production of pain and its sensory pathways from the heart, the operation should be performed only on patients who suffer intensely, who have not improved under well-directed medical care and who, in the opinion of the physician, will stand surgical therapy without undue risk. These patients should be thoroughly studied both before and after sympathectomy. Detailed follow-up records, kept

over a period of years, will afford a basis for judgment as to its value. It is my present opinion that this operation will prove to have a very limited field of usefulness.

The paravertebral injection of alcohol into the posterior nerve roots has recently been advocated.[21] It is an uncertain procedure, unaided by visual guidance, and not without hazard. Richardson and White,[22] on the basis of a limited experience, have expressed the belief that alcohol injection is superior to sympathectomy. The relief afforded in some instances may well be due to blocking of cutaneous impulses, analogous to the results of skin infiltration with novocaine in the areas of cutaneous hyperalgesia, as practiced by Weiss and Davis.[23] Such cutaneous injections give prompt but only temporary relief in certain cases.

SUMMARY

The "disorder of the breast" described by Heberden, in the light of increasing experience, has proved to be the symptomatic manifestation of many pathological states. Perpetuation of the name originally given to the condition, and the concept of angina as a clinical entity, has resulted in confusion and disagreement as to its precise meaning. It is, therefore, suggested that the term "angina pectoris" be abandoned. Correlation of clinical and pathological data has demonstrated that cardiac pain may be associated with a variety of structural and functional changes. Pain resulting from disturbances in the region of the heart is best described as *cardiac pain*. In making a complete cardiac diagnosis, this should be qualified by a statement as to the probable structural and functional changes with which the pain is associated. Further knowledge concerning the mechanism of pain production may point the way to a more precise terminology. The conception of pain as a symptom will make for better diagnosis, for rational therapy, and for more accurate prognosis.

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PAIN DUE TO TEMPORARY OCCLUSION OF THE CORONARY ARTERIES IN DOGS*

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PORTER[1] stated that ligation of the coronary arteries in the dog did not produce pain. Smith also failed to observe pain in similar preparations. We do not believe, as do these investigators, that these observations offer convincing evidence against the coronary theory of pain. The occlusion was produced under anesthesia. Following severe thoracic surgery, an animal is depressed for hours and shows little evidence of pain that he may be suffering. Moreover, the pain of coronary occlusion may be transient. Until sudden occlusion of the coronary arteries has been produced in the conscious dog, one cannot conclude that the process is without pain.

Two years ago we began to study the effects of sudden coronary occlusion in conscious dogs. Observations made upon the pain response are herewith presented.

METHODS

The operation was performed under ether anesthesia by intratracheal insufflation (Meltzer[5]) and no other medication. The chest was opened by a transverse incision in the third or fourth intercostal space, carried from one side of the chest to the other, dividing the sternum. A rib spreader was inserted and a splendid exposure obtained. The pericardium was opened freely and the coronary arteries were exposed by dissection at the base of the heart. A thread of vaselined heavy linen was passed under each coronary artery within a centimeter of its exit from the aorta. In experiments where only the branches of the left coronary artery were to be studied, separate threads were passed under the vessels immediately after the bifurcation. The threads were attached to special devices for occluding the vessels and the chest was closed securely, after taking care to avoid partial pneumothorax. The devices for vessel occlusion were of two types. In some experiments the coronary traction threads were fastened to the end of a specially prepared camera shutter release, which protruded from the chest at the wound and was arranged in such a manner that by pressing the release, a short bar, through which the thread had been led, pressed firmly upon the artery. This method prevented interference with the heart itself, as would occur with direct pull upon the coronary vessel. A second method was that of

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attaching the traction threads to a suitable wire which was introduced into the thoracic cavity from a stab incision in the base of the neck. Traction upon the wire exerted traction upon and occlusion of the coronary vessels with minimal disturbance to the heart because of the axial direction of the pull. By either method the arteries could be occluded or released at will. In our earlier experiments we adhered diligently to asepsis but later abandoned it as unnecessary, since the animals were in good condition for experimentation within a few hours after operation. Three to four hours after the operation pain responses were studied. In some animals the pain response was so marked as to interfere with the electrocardiographic curves and morphine was given for this part of the experiment.

RESULTS

Fifty dogs were used. Ten were not fully conscious at the time coronary occlusion was produced and pain response could not be depended upon. Of the remainder the following examples are typical:

Protocol.—Mongrel dog, medium size. A traction thread was placed on the left anterior coronary artery immediately below its bifurcation and attached to a shutter release. Three hours later the animal was fully awake and lying quietly with E. K. G. leads attached. Vessel was suddenly occluded. Dog immediately howled and struggled. The vessel was released and dog became quiet at once. Repetition twice obtained the same results. Morphine phosphate, gr. ⅛, was given by hypodermic injection. Fifteen minutes later coronary artery was again occluded. Dog howled less vigorously and moved left fore-leg and paw. The vessel was released again with cessation of pain response. Repetition procured the same effect.

Protocol.—Mongrel dog of medium size. Traction threads were placed around each coronary artery near its origin and attached to the wire device. Four hours later the dog was fully awake and was connected to the E. K. G. leads. Gentle traction on left coronary artery. Dog uttered sharp cries, struggled, and attempted to arise. After 25 seconds vessel was released and animal became quiet at once Gentle traction on right ligature obtained the same response. Several repetitions. Occlusion, then, of both vessels simultaneously produced the same type of symptoms but more violent. After two minutes of occlusion of both vessels ventricular fibrillation began, the pain response ceased, and death occurred from heart failure.

Most of the dogs responded similarly to these. In the majority of cases in which both coronaries were occluded simultaneously the heart did not fibrillate. Some of the dogs were less active and responded to occlusion by whimpering and weakly "purposeful escape movements." Body movements due to pain were easily distinguished from others, especially from asphyxial convulsions and other automatic responses, by their purposeful nature. The ten dogs that gave less clear-cut responses had survived the operation poorly or had been kept too long before being tested.

Electrocardiograms of all animals were taken and will be reported later.

SUMMARY AND CONCLUSIONS

It is pointed out that previous investigators have failed to get pain response in dogs following coronary occlusion, possibly because the animals had adjusted themselves to the altered cardiac condition by the time they recovered sufficiently from the anesthetic and surgical insult.

The detection of pain in these experiments was made possible by avoidance of anesthesia at the time of coronary occlusion. Acute severe pain was produced in forty out of fifty dogs by sudden occlusion of either coronary artery or of either or both branches of the left coronary artery, the pain beginning almost instantly after occlusion, lasting throughout occlusion, and ceasing immediately with release of the occlusion. When both coronary arteries were occluded simultaneously, similar responses resulted except that in a few cases ventricular fibrillation and death resulted. The ten dogs that gave less clear-cut results were not sufficiently alert to respond properly.

The responses are due to real pain and not to cardiocirculatory failure and asphyxia.

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THE RELATION OF CLINICAL, INCLUDING ELECTROCARDIOGRAPHIC, PHENOMENA TO OCCLUSION OF THE CORONARY ARTERIES BASED ON THE OBSERVATION OF A CASE*

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THE nature and origin of cardiac pain are still unsettled. The first contribution on this subject, made by Heberden[1] in 1768, emphasized, in describing the symptom complex which he called "angina pectoris," as the outstanding phenomenon "a painful and most disagreeable sensation in the breast" which affects persons while "walking and more particularly when they walk soon after eating." It does not cause breathlessness and stops on standing still. Sudden death was its other feature. Heberden saw no autopsies and refrained from associating these phenomena with any lesion. In 1776, Edward Jenner, in a letter to Heberden regarding John Hunter[2] and in another to Parry,[3] first suggested the association of angina pectoris with disease of the coronary arteries. He came upon this notion from post-mortem examinations of the hearts of persons who had been subject to such attacks. By 1799, Parry,[3] although he discussed the relation of anginal attacks to exertion, did not think that exertion was a necessary part of the symptom complex and did not include it in the formulation of his definition of angina pectoris. Nor is mention made one hundred years later of exertion in the "definition" of angina pectoris in Romberg's,[4] Strümpell's[5] or Osler's[6] textbooks. Allbutt[7] has this to say: "That the moments of the several seizures are often determined by effort, even by effort of mind or emotion, we know only too well; so well indeed that there is a notion abroad—one which more than once has been mentioned to me in discussing a diagnosis—that attacks are always so determined. Huchard countenanced this erroneous notion. Yet from Heberden himself we have learned that attacks may occur during sleep, often during rest."

In the course of the century a change in the meaning of the term originally proposed by Heberden has obviously taken place. In attempting to understand the history of ideas such as this, it would be interesting to learn how this change came about, but we have not searched the literature to trace the sequence.

Herrick[8] in 1912 gave the first comprehensive statement of the clinical features exhibited by one class of patients who are subject to pain and in whom occlusion of a branch or branches of the coronary artery

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is the etiological factor. In spite of the accumulation of a vast literature on this subject since then, the pathogenesis is still far from clear. The minimum signs and symptoms upon which one may venture to make a diagnosis of coronary occlusion and to give and to estimate the state of the coronary vessels and of the heart muscle are not known. Nor is it clear how much stress to place upon cardiac pain alone in deciding whether a coronary accident has occurred or whether pain is caused by a more transient process, such as spasm. It is in order to contribute to this phase of the matter that the case of this patient is published. For this purpose his clinical course was sufficiently well observed and the pathological examinations at autopsy were sufficiently well made.

CASE REPORT

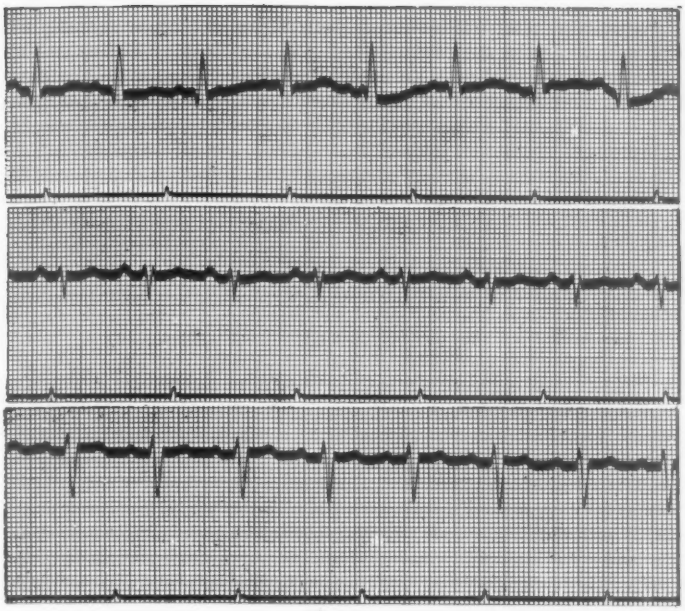
H. R., Hospital No. 4734, was a male, sixty-seven years old. He was admitted to the Rockefeller Hospital on April 4, 1923, having complained of shortness of breath for from eight to twelve months, swelling of the legs for eight months, tenderness in the region of the liver for from eight to twelve months and cough for one week.

The family history was negative.

Past History.—There was no history of acute rheumatic fever, syphilis, or any other diseases having cardiac sequelae. He had enjoyed excellent health until the onset of the present illness.

Cardiac History.—In August, 1919, the patient was awakened at night with severe pain over the chest in front. He was so ill that he was unable to remember the character of the pain. He was too ill to be moved at once, but was taken to a hospital the following day. The pain continued for twenty-four hours. After four or five days 1000 c.c. of fluid were removed from the right pleural cavity by paracentesis. Following this treatment he improved and was discharged in two weeks. He felt weak and was unable to do what he wished. A year before admission to the Rockefeller Hospital he became short of breath on walking. Dyspnea gradually increased until he was forced to discontinue climbing stairs. Eight months before admission edema of the feet and legs first appeared. Shortly afterward tenderness in the right upper quadrant of the abdomen developed. Two months later he began to sleep on three pillows. Gradually all his symptoms, that is to say, dyspnea, edema, and tenderness of the liver, increased. From the onset of cardiac symptoms he received digitalis irregularly. The drug had not been given for several months.

Physical Examination.—The patient was an undernourished white man who lay propped up in bed and was orthopneic. Dyspnea was very marked when he attempted to lie flat. There was slight cyanosis of the lips. The temperature (rectal) was 100.7° F., and respirations 30 per minute. A tracheal tug was not felt. The heart was enlarged. The rhythm was regular, the rate 96 per minute. There were no thrills and no shocks over the precordium. The sounds over the base were clear. At the apex a soft blowing systolic murmur replaced the first sound. The murmur was not transmitted to the axilla. The radial vessels were thickened but were not tortuous. The systolic blood pressure was 130 mm. mercury and the diastolic, 85 mm. There was no capillary pulsation in the finger tips. Clubbing of the fingers was not present. There were signs of fluid in both pleural cavities. The liver was tender and was felt to within one finger's breadth of the umbilicus. There was pitting edema of the feet, ankles, and legs as far as the knees.



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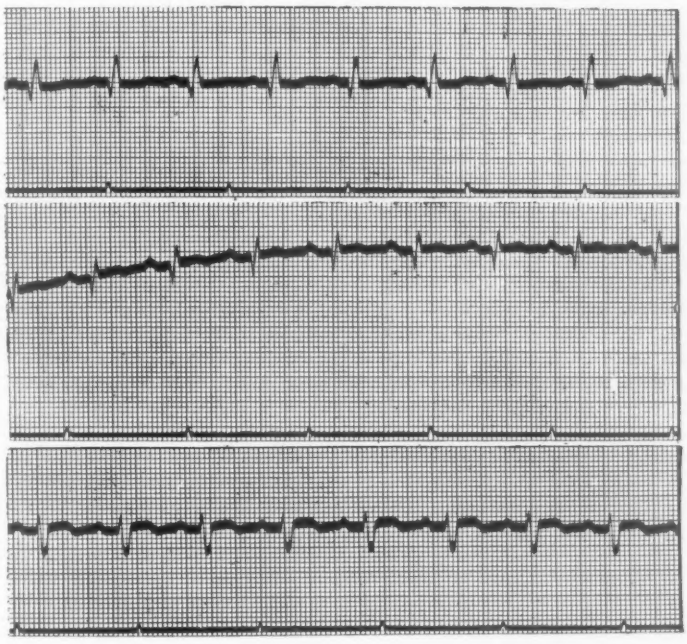

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Fig. 1.—The three standard leads of the electrocardiogram are reproduced. The electrocardiograms from above downward read Leads I, II, and III. Divisions of the ordinates equal 10^{-4} volts. Divisions of the abscissae equal 0.04 of a second. The signal at the bottom of each record indicates seconds. The electrocardiograms are reduced to two-thirds of their natural size. Fig. 1*a* is a photograph of an electrocardiogram made on April 6, 1923. This electrocardiogram is typical of the form exhibited before the occurrence of the attack of cardiac pain on April 10, 1923. In Fig. 1*b* is reproduced the electrocardiogram taken on April 10, 1923, several hours after the attack. All the electrocardiograms taken afterward resembled this one. Not only are there changes in the form of the R- and S-waves, but the waves are of lower voltage.

The electrocardiogram showed a normal sinus rhythm. The conduction time was normal and there was left ventricular preponderance.

The count of the red blood cells was 5,000,000. The hemoglobin (Sahli) was 97 per cent. The count of the white blood cells was 11,600, 65 per cent of which were polymorphonuclear in form. The phenolsulphonephthalein excretion was 65 per cent. The urea concentration index (Van Slyke index) was 36.7. The Wassermann reaction in the blood was negative. There was a trace of albumin in the urine; in the sediment there were many leucocytes and epithelial cells.

Course in the Hospital.—During the first few days the edema decreased.

On April 10, 1923, he experienced severe precordial pain during the night. At 9 A. M. premature contractions were occurring every fourth beat. Occasionally during auscultation there were runs of premature contractions, but when electrodes were applied, the irregularity was no longer present. The electrocardiogram showed splitting of the S-wave in Lead III (Fig. 1*b*). This wave had been sharply spiked in all the previous records (Fig. 1*a*). The voltage of the QRS complexes decreased. The conduction time increased from 0.16 to 0.18 of a second to from 0.20 to 0.21 of a second. There was no friction rub and no rise in temperature.

On April 13, the patient received digitan (Merck) 2.0 gm. by mouth within twenty-four hours. The ventricular rate slowed to from 70 to 80 per minute from having been between 90 and 100. The electrocardiogram showed further changes in the T-waves but no change in P-R time. There was no increase in the output of urine.

On April 18, 890 c.c. of fluid were removed from the right pleural cavity. Dyspnea was somewhat relieved. On April 19, during the morning, 610 c.c. of fluid were removed from the left pleural cavity. At 3:30 P. M. the patient was sitting in bed talking to his wife. Suddenly he fell back against the pillows gasping for breath. There was marked cyanosis. The ventricular rate was from 35 to 40 per minute and the rhythm seemed to be regular. There was no pulse deficit. He was unable to speak. Respirations became slower and more labored; cyanosis increased. The patient died within five minutes of the onset.

Summary.—The patient was admitted to hospital with fairly marked heart failure. In the absence of a history of acute rheumatic fever and of syphilis (the Wassermann reaction was negative) and in the absence of hypertension or of valvular disease on examination, we were of the opinion that senile changes had taken place in the heart muscle accompanying changes in the arteries. He had suffered from only two attacks of cardiac pain. The first attack occurred three and a half years and the second nine days before death. *Both attacks occurred at night while the patient was sleeping,* that is to say, they were not associated with exertion. Electrocardiographic data are not available for the first attack of pain; the second attack, however, was followed by certain changes (notching and decrease in amplitude) in the ventricular complexes of the electrocardiogram. Fluid was removed from both pleural cavities. Digitalis slowed the ventricular rate and gave characteristic changes in the T-waves of the electrocardiogram. There was no diuresis, however, following its administration. The patient was apparently slowly improving when he suddenly died. The diagnosis* was: *Etiological:* arteriosclerosis; *Anatomical:* cardiac hypertrophy, myocardial degeneration, coronary thrombosis, left ventricular preponderance; *Physiological:* normal sinus rhythm, relative mitral insufficiency, congestive heart failure.

SUMMARY OF AUTOPSY FINDINGS

The autopsy was performed by Dr. Arnold Branch an hour and a half post-mortem.

*This diagnosis conforms to the nomenclature for cardiac diagnosis approved by the American Heart Association. AM. HEART J. 2: 202, 1926-27.

Anatomical Diagnosis.—Extreme thrombosis and atherosclerosis of the left coronary artery, focal thrombosis and atherosclerosis of the right coronary artery, myocardial degeneration, healed and recent myocardial infarcts, adherent pericardium, fibrous pleurisy, anthracosis, emphysema, arteriosclerosis, venous stasis of the liver, perisplenitis and perihepatitis, abdominal adhesions, hyperplasia of the spleen, arteriosclerosis of the kidneys, infarcts of the kidneys.

Gross Examination.—The body was that of a well-developed white man. The skin was pale and dry.

Thorax.—The lungs were voluminous. The precordial area was large. The right pleural cavity was almost completely obliterated by firm fibrous adhesions. The left pleural cavity was free except for fibrous adhesions in a small area laterally and posteriorly. There were a few bands between the left border of the pericardium and the lung, involving both the visceral and parietal layers of the pleura.

Lungs.—The lungs were moderately anthracotic and emphysematous. On section they were pale and spongy; they showed no gross lesion. The peribronchial glands were moderately enlarged and anthracotic. There was a moderate degree of atheromatous thickening in the larger branches of the pulmonary artery.

Pericardium.—There was a moderate amount of fat around the parietal pericardium. This layer was attached everywhere to the visceral layer by loosely adherent fine fibrous tissue. When the parietal layer was stripped back, it exposed a purplish brown surface over which were scattered small hemorrhagic areas. These were especially clustered around blood vessels.

The *heart* weighed 530 gm. The wall of the *left ventricle* measured 16 mm. in thickness at the base, 15 mm. at the level of the papillary muscle, and 4 mm. at the apex. The muscle was brownish-red and firm. The cavity was large, especially at the apex, the muscle there being thinned out and scarred. This thinning occurred abruptly. The papillary muscles were slender. The endocardium, in which there were scattered yellow patches of thickened endothelium, was pale brown. Scarring of the deeper layers produced a mottled appearance. One dark purple area about 3 cm. in diameter (Fig. 2*a*) occupied the base of the posterior papillary muscle. It was probably a recent infarct. The yellow scars occurred in greatest number in the apex, in the interventricular septum, under the aortic valve, and at the junction of the chordae tendinae with the papillary muscle (Fig. 2*a*). By far the largest irregular yellow area (3 cm. by 3 cm.) was situated partly in the anterior wall and partly in the septum midway between the apex and the base and just below the large left anterior descending branch of the coronary artery and differed from the other areas in that on cutting it definite scars were seen in the underlying muscle, the yellowish appearance not being due merely to thickened endocardium.

The wall of the right ventricle measured 6 mm. in thickness at the base, 7 mm. at the level of the papillary muscle, and 4 mm. at the apex. The muscle was firm and brown, but there was more epicardial fat than on the left ventricle. The papillary muscle and the cavity were not large. The endocardium was thickened in only three small places, two being on the anterior wall and the third on the septum.

The endocardium of the *left auricle* was slightly but definitely thickened and wrinkled. The wall measured 4 mm. in thickness. The cavity was not enlarged.

The endocardium of the *right auricle* was thickened around the orifices of the venae cavae. It was smooth throughout. The thickness of the wall was from 2 to 3 mm. The cavity was not enlarged.

Mitral Valve.—The mitral ring measured 8.5 cm. No stenosis was present. The lateral cusp was slightly thickened only at the line of closure. The aortic cusp showed yellow thickening at the base and more marked thickening near the free edge with a narrow transparent zone between. It was covered with endothelium

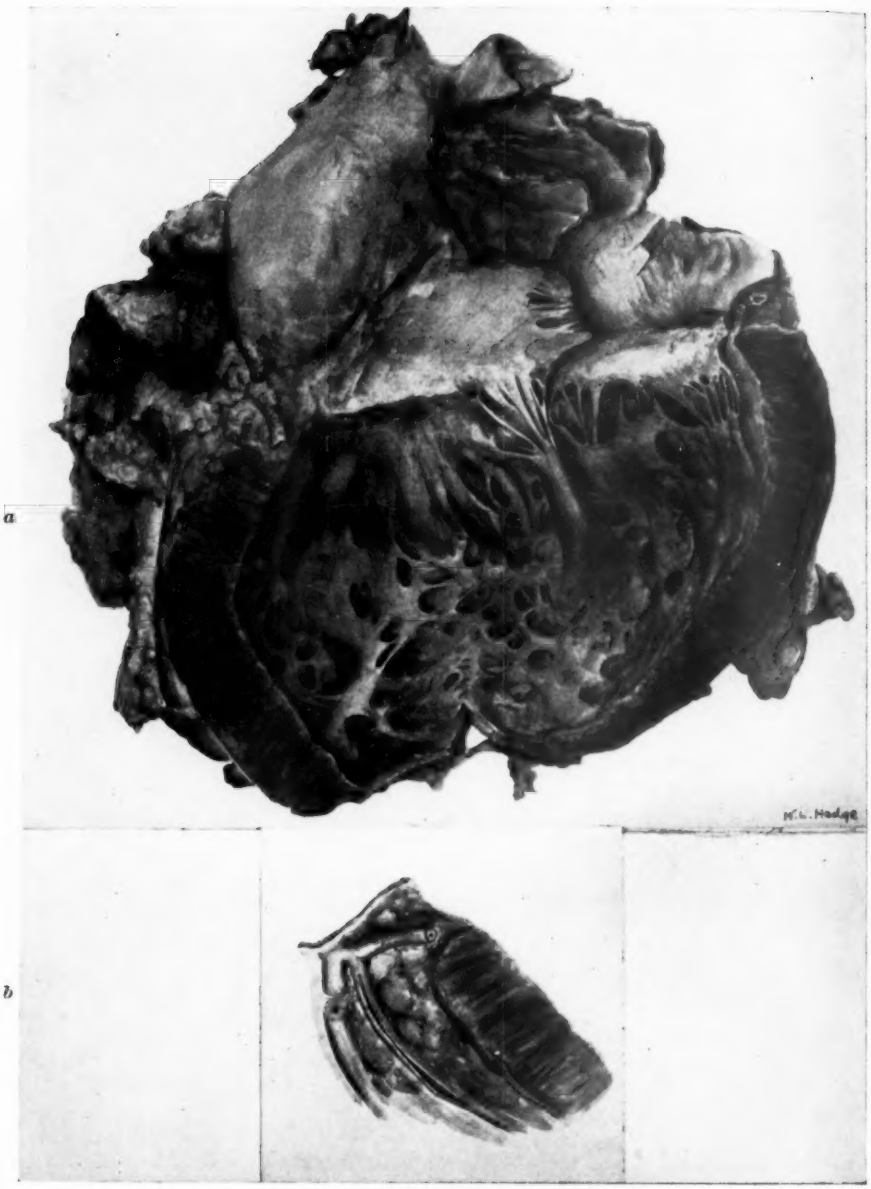

Fig. 2.—In Fig. 2*a* is presented a photograph of a colored drawing of the cavity of the left ventricle. A recent infarct in the regions of the posterior papillary muscle is readily identified by the dark area. The whitish areas are due to thickening of the endocardium. Scarring at the apex and thinning of the muscle here are to be noted. In Fig. 2*b* is shown a photograph of a colored drawing of the left coronary artery and its branches.

throughout. Along the line of closure, which was but slightly thickened, there was a reddish discoloration. A series of tiny nodules occurred at the junction of the chordae tendineae.

The leaflets of the *tricuspid valve* were mobile. The ring measured 12.7 cm. There was no thickening.

The *aortic valve* was well preserved; there was only slight thickening of the endocardium in the right posterior cusp and on the interventricular septum below the valve at its junction with aortic cusp of the mitral. Nowhere was there calcification. The opening measured 5.4 cm. in circumference.

There was no abnormality of the *pulmonary valve*. It measured 6.6 cm.

At its origin the *right coronary artery* showed definite hard thickening for a few millimeters of its length, but the vessel was patent. It then appeared normal for a short distance. At the point where the first noticeable branch to the auricles and pacemaking area was encountered there was a marked yellow thickening; the orifice of this small artery was narrowed, but in the rest of its extent, as far as it can be traced to its finer terminations, the walls were thin and elastic and the lumen was patent. The whole of the *right circumflex artery* appeared normal with the exception of a few scattered small yellow plaques. The *posterior descending branch* was normal until a point was reached in its course down the posterior wall of the left ventricle, just above its final bifurcation into the two terminal arteries. At that point the wall was thickened, firm, calcareous, irregular and bead-like, and the lumen could not be seen. Below this occlusion, which was about 0.5 cm. in length, the artery again appeared normal. The lesions just described appeared to be old, but it is quite possible that a very small recent clot might have sufficed completely to block it.

The *left coronary artery* (Fig. 2*b*) immediately divided into three branches, namely, the large *left anterior descending branch*, a fairly large *marginal branch*, and a quite well-developed *left circumflex branch*. At its orifice it was thickened, but the lumen was patent. The wall of the left anterior descending branch was so firmly calcified that it was cut only with difficulty; the lumen was apparently blocked throughout by a yellow gelatinous clot. This artery lay over the area of extensive scarring seen on the internal aspect of the left ventricle. The *marginal branch* was completely blocked about 1.5 cm. from its origin by a reddish thrombus; the artery after this point was very firm and thickened. Below this it again became patent and appeared normal. The *left circumflex branch* was blocked by a red thrombus. The walls were thickened until it branched into smaller arteries on the posterior surface. These smaller branches appeared normal.

The *aorta* was slightly thickened in the sinuses of Valsalva and arornd the coronary openings. In the arch and in the lower abdominal aorta there were fairly marked atherosclerotic changes. In the thoracic portion these occurred as localized thickenings covered by endothelium which were especially marked around the orifices of the intercostal vessels. At the lower end, however, there were plaques of calcification and ulceration.

The larger branches of the *pulmonary artery* in the lung showed a slight degree of sclerosis.

The *spleen* weighed 110 gm. In the capsule which was attached to the diaphragm by fibrous adhesions were localized thick yellow plaques. There were no infarcts to be seen.

The *adrenals* were well preserved and large. The pancreas was pinkish-gray in color.

The *left kidney* weighed 130 gm. The capsule could be stripped with slight difficulty. On section the cortex was wide; the medulla, pale. Differentiation of cortex and medulla was not sharp, but the kidney appeared very well preserved. In the cortex at the lateral border was a small firm yellow area of infarction.

The pelvis and ureter were intact. The *right kidney* weighed 120 gm. It was similar to the left, except that there were no infarcts.

The *liver* weighed 1320 gm. The capsule appeared thickened along the inferior surface of the anterior border. It was yellowish-red and mottled. On section it exuded blood. The markings were fairly distinct; the substance was friable.

On section of the *brain* there were no areas of hemorrhage to be seen, and the blood vessels at the base showed no evidence of atherosclerosis.

MICROSCOPICAL EXAMINATION

Coronary Arteries.—Sections were examined from the main branches of the left coronary artery, that is to say, the left anterior descending, the left marginal, and the left circumflex. The lesions in all were identical. The adventitia was intact, the media narrow, and the intima thickened. The elastic tissue stain showed this quite definitely. The lumen was obliterated by healed canalized fibrous tissue in which there were a few pigment-containing cells but no inflammatory exudate (Fig. 3). The areas of canalization were relatively small in comparison with the fibrous tissue and in no wise approached the cross-section of the lumen of the vessel if it had been normal. These small vessels varied in size from capillary diameter to that of small arterioles. They numbered from five to over a dozen in the various sections. A few contained red blood cells and appeared patent while the majority were filled with pink staining, homogeneous hyaline-like material, not taking the characteristic fibrin stain. In a few this pink tissue formed a thick ring around the lumen. One could imagine that only a few were patent. This appearance was due to healed canalized thrombi; there may have been recent thrombosis.

Left Ventricle.—In none of the sections was there evidence of active inflammation. The section from the posterior wall showed areas of fibrous scarring. The muscle fibers were large and contained large nuclei; distinct striations and contractile elements were present. In a section from the region of the anterior papillary muscle a similar condition was observed. In the septal area definite discrete fibrous intimal thickening was noted, the tissue being relatively acellular. The posterior papillary muscle was the locus of a recent infarct (Fig. 2*a*); the musculature was infiltrated with red blood cells and a few white blood cells. The muscle tissue here too appeared rather homogeneous and striations were less distinct. In a section from the apex the wall was thin (Fig. 2*a*), and the pericardium was thickened and adherent. The endocardium was thickened, and there was marked fibrosis and replacement of the muscle tissue. There was in short, endocardial thickening, fibrosis, and recent infarction. A section from the lateral wall of the *right ventricle* showed no change from the normal.

The endocardium of the *left auricle* was slightly thickened. One vessel showed evidence of fibrous thickening. There was definite thickening, fusion, and hyalinization of the pericardium of the *right auricle;* there was sparse perivascular small round cell infiltration in the parietal layer.

Aorta.—The adventitia was normal, as was also the media. The intima was irregularily thickened and deep in its substance were areas made up of pale staining acellular necrotic tissue. The section included the origin of a small vessel, and at this point blood appeared to have welled into a space in the intima raising the surface layers. At no place was there ulceration. The appearance was that of atherosclerosis.

The *medullary* vessels of the *kidneys* were engorged as were also the glomerular tufts. The vessels were rather thickened; there was an occasional tuft with perivascular fibrous thickening. Small infarcts were seen in the cortex just under the capsule; these were infiltrated with small round cells. In infarcts which were larger but still microscopical in size there were whole areas of tubular and glomer-

ular tissue which had undergone necrosis. The vessels in the vicinity were congested; around the thrombosed vessels causing the infarct there was hemorrhagic extravasation. In Regaud-fixed tissue mitochandria had disappeared. There were present then infarction and atherosclerosis (slight).

Liver.—There was early cyanotic atrophy. The capsule was slightly thickened, and there was subcapsular fibrosis. The appearance was that of venous stasis.

Lungs.—There was fibrous thickening of the pleura and anthracosis. There were pigmented mononuclear phagocytes in the alveoli.

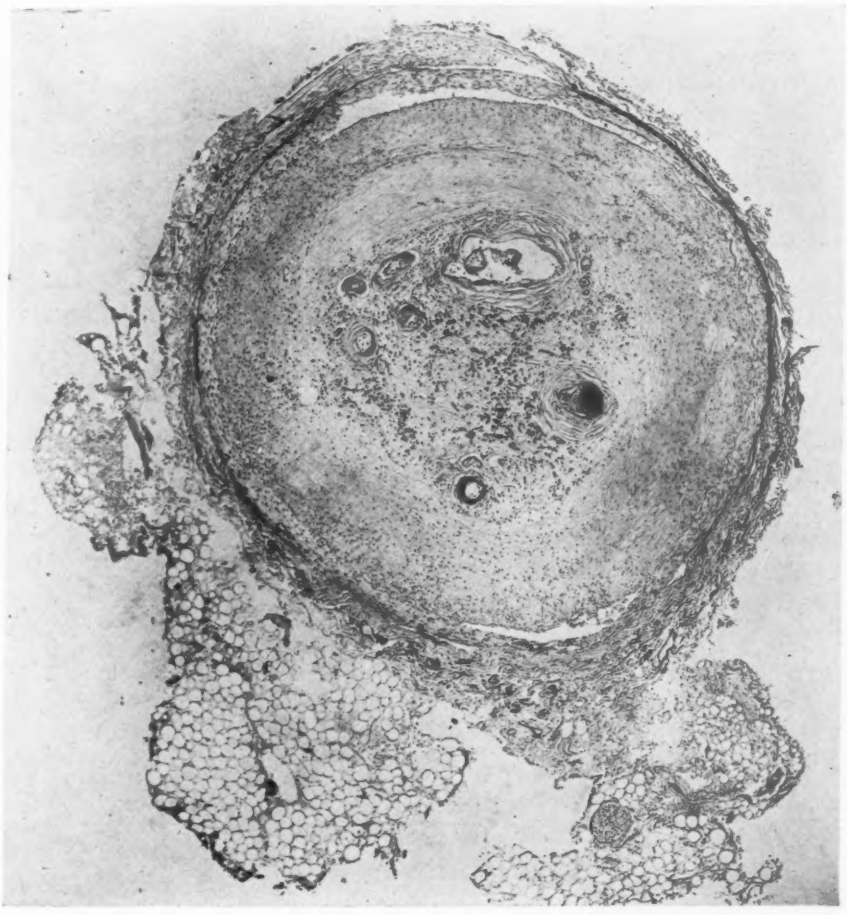

Fig. 3.—In this figure is reproduced a microphotograph of a cross-section of a branch of the left coronary artery. For description see text. Magnification 42. (? reduced.)

Spleen.—There was perisplenitis. The capsule was infiltrated with cells, the sinuses were filled with blood, the pulp was not hyperplastic. The blood vessels were thickened. The malpighian corpuscles were numerous.

Summary.—The chief interest at autopsy was found to lie in the heart. Both healed and more recent infarcts of the left ventricle and septum were found. The apex was very thin and scarred. In the coronary vessels supplying the left ventricle were areas of calcification and thickening, and on section the lumen of the

vessels appeared to be completely closed. The healed thrombi were pierced by small canalized vessels. The area of scarring in the heart muscle corresponded to the areas supplied by the vessels which were occluded by the canalized thrombi.

DISCUSSION

The clinical and post-mortem findings of a patient dying of coronary occlusion have been presented. From examination of the heart at autopsy there is no doubt that the patient had suffered from infarcts in the heart on many occasions. These had apparently occurred without causing conspicuous symptoms. There was nothing in the history, physical examination, or electrocardiographic record that led to the suspicion that the patient had been the subject of such grave changes in the myocardium and coronary vessels as were found at autopsy. There were only two attacks of cardiac pain; the first occurred three and a half years before admission to this hospital, and the second nine days before death. Both of these attacks came on at night while the patient was asleep. They occurred obviously *without physical exertion.* Following the last attack of severe cardiac pain, there occurred the only disturbance of cardiac rhythm of which we have knowledge. There were none of the usual signs associated with coronary thrombosis, such as rise in temperature or pericardial friction rub. The only indication appropriate to the lesion which the autopsy disclosed was the count of the white blood cells which rose from 11,600 (April 8) to 16,400 (April 12) and fell later to 10,800 (April 14). According to Wearn,[9] however, the classical signs of coronary occlusion may not appear when it occurs in the presence of heart failure. Friction rub may have been absent because the pericardium was adherent. At autopsy there was a fresh infarct in the left ventricle, at the base of the posterior papillary muscle corresponding to a region supplied by the circumflex branch of the left coronary artery. The cardiac pain from which the patient suffered nine days before death may have been due to the occurrence of this infarct. On the other hand it may have been of more recent origin. Evidence that damage to the cardiac muscle had occurred after the time of the cardiac pain on April 10, was shown in the electrocardiogram (Figs. 1*a* and 1*b*) by splitting the S-wave in Lead III. There are, of course, corresponding changes in Lead II. Here the R-wave was followed by a somewhat deeper S-wave. After the attack of cardiac pain the sequence of these waves was, however, reversed (Fig. 1*b*) and the ventricular complex was opened by a negative followed by an approximately equal positive swing. These changes indicated without a doubt change in the path of the excitation wave. There occurred also decreases in amplitude of the QRS complexes (compare Figs. 1*a* and 1*b*). Smith[10] has called attention to this occurrence following occlusion of the left coronary artery, especially when the circumflex branch is involved either alone or with the ante-

rior descending branch. According to this observer this change is found with less frequency, however, than are alterations in the T-wave. None of the changes in the T-wave, such as have been described by Herrick[11] and by Pardee,[12] were seen. The autopsy, nevertheless, revealed lesions in branches of the left coronary artery which, according to the experiments of Smith and reports of cases by Herrick, Pardee and others, are the occasion of such changes in the electrocardiogram. The lesions were due as has been stated to extensive arteriosclerotic changes. All three branches of the left coronary artery were profoundly involved, a recent infarct occurring in a region supplied by the circumflex branch, while the right one was less affected. In all of them there were old thrombi which had become canalized. In some, however, the thrombi were of more recent origin. It is difficult to see how an adequate blood supply to the heart muscle was maintained under these circumstances. The heart muscle can, of course, have been nourished by way of the thebesian vessels. Wearn,[13] in the light of recent studies, thought that in the event of gradual closure of the orifices of the coronary arteries, the thebesian vessels can supply the heart with sufficient blood to enable it to maintain an adequate circulation. It is impossible to decide whether death was due to gradual closing off of the blood supply by thickening of the intima and thrombus formation until a state was finally reached where the supply of blood was inadequate, or to sudden complete blocking of the circumflex branch of the left coronary artery which caused the infarct in the region of the posterior papillary muscle. If, however, the infarct occurred nine days before death and was the occasion of the cardiac pain experienced by the patient at that time, it is possible to connect the occlusion of the circumflex branch of the left coronary artery with the electrocardiograph changes which were then observed. No attempt has yet been successful, however, in correlating specific changes in electrocardiogram with injury to special coronary vessels. The time has come, perhaps, when an effort should be made to recognize certain distinctions. It is to be expected that when occlusion (and by this is meant the acute lesions, not the slowly developing senescent process*) takes place, fever, leucocytosis, and pain occur, irrespective of the branch of the coronary artery which is involved. To identify the branch that is occluded, a relation should be shown to exist between the change in the electrocardiogram and pericardial friction rub on the one hand and the exact vessel occluded on the other. In examining the reports of the published cases it has not been possible in most instances to obtain such information in order that it might be arranged in a way to correlate sign and pathology. In searching the literature, as has been said, only those cases have been sought in which an acute

*How the slowly developing senescent process terminates is scarcely known. It may include a transition between gradual narrowing and sudden occlusion, when a condition simulating an acute lesion may appear.

TABLE I

SUMMARY OF CASES IN WHICH OCCLUSION OF A CORONARY ARTERY WAS DEMONSTRATED AT AUTOPSY EXAMINATION AND IN WHICH ELECTROCARDIOGRAPHIC REPORTS ARE AVAILABLE

AUTHOR	REFERENCE	PATIENTS	AGE IN YEARS	EXPERIMENTS	SYMPTOMS	EXERTION AS EXCITING CAUSE	SIGNS	COEXISTING ELECTROCARDIOGRAPHIC CHANGES		CORONARY ARTERY FOUND TO BE INVOLVED AT AUTOPSY EXAMINATION
								DECREASE QRS	T-WAVE NEGATIVE	
Herrick	J. A. M. A. **72:** 387, 1919	Case 3	42		Substernal pain, tachycardia	Not known	Not given	+ (178 days after occlusion)	+ (T_2 and T_3 41 days after occlusion)	Descending branch of left coronary artery; also the circumflex branch by old thrombi
Smith	Arch. Int. Med. **32:** 497, 1923	Case 1	44		Stab wound				+ (17 days after ligation)	Ligation of descending branch of left coronary
		Case 3	51		Substernal pain	0	Not given	+ (11 days after pain)	+ (11 days after pain)	Descending branch of left coronary and also two branches of the circumflex branch; died 11 days after pain
		Case 5	39		Pain, nausea and vomiting	?	Friction rub	+ (2 weeks after onset)	+ (2 weeks after onset)	Descending branch of left coronary; died 2½ months after pain, infarct healed
Herrmann	J. Missouri M. A. **17:** 406, 1920	Case 2	61		Pain	+	Friction rub, fever, leucocytosis	+		Thrombosis of descending branch of left coronary
		Case 6	53		Pain	0		+		Thrombosis of descending branch of left coronary

TABLE I—CONTINUED

AUTHOR	REFERENCE	PATIENTS	AGE IN YEARS	EXPERIMENTS	SYMPTOMS	EXERTION AS EXCITING CAUSE	SIGNS	COEXISTING ELECTROCARDIOGRAPHIC CHANGES		CORONARY ARTERY FOUND TO BE INVOLVED AT AUTOPSY EXAMINATION
								DECREASE QRS	T-WAVE NEGATIVE	
Stewart	Case now being reported		67		Pain	0	Leucocytosis (no fever and friction rub)	+	0	Fresh infarct in area supplied by circumflex of left coronary artery which was occluded; died 9 days after onset of pain and Ekg. changes
Smith*	Arch. Int. Med. 22: 8, 1918			56 dogs 8 dogs					+ 0	Any branch of left Any branch of right
Smith*	Arch. Int. Med. 25: 673, 1920			14 dogs 5 dogs					+ 0	Descending branch of left and circumflex branch Right coronary
Smith*	Arch. Int. Med. 32: 497, 1923			records of 29 dogs of 1918 series (see above) restudied				+ in 100 per cent + in 20 per cent + in 30 per cent	According to 1918 report T-waves negative in all instances	Left Circumflex branch Descending branch of left coronary Left circumflex as well as descending branch of left coronary

*In the case of the experiments reported by Smith it is the artery which was ligated that is referred to in the last column.
+ = present
0 = absent

lesion was followed in turn by changes in electrocardiogram and then within a short time by autopsy examination. It is only in this way that specific electrocardiographic signs can be correlated with lesions in special coronary vessels (Table I).

Smith[10] found in dogs decreases in amplitude of the QRS complexes in all the cases when the circumflex branch of the left coronary artery was occluded alone, in 20 per cent when the anterior descending branch was occluded alone, and in 30 per cent when the anterior descending branch as well as the circumflex branch was involved (Table I). He cites 2 cases (Herrick's[11] and his own) in which comparable electrocardiographic signs were observed when there was occlusion of the left circumflex branch and also of the anterior descending branches, though the thrombi in the circumflex branches were of more recent origin. Pardee's case[12] unfortunately cannot be made to serve as evidence, since the patient recovered and the vessel involved is not known. His other published cases[14] cannot be utilized in this connection, since the patients died of heart failure and did not exhibit signs or symptoms associated with disease of the coronary arteries, although at autopsy alterations were found in these vessels. So far as can be ascertained death did not follow acute coronary occlusion. It is not possible in Wearn's 19 cases[9] to correlate the vessels involved with the electrocardiographic changes. He states, however, that the QRS complex was diminished in height in about one half, while in only one of them was the circumflex branch occluded. In the 2 cases of thrombosis of the descending branches of the left coronary arteries reported by Herrmann[15] the QRS complexes were diminished in height. It is not known whether the T-waves changed. In Willius'[16] cases the changes in the coronary vessels were of a chronic nature, and the cases do not fall in the group under consideration. In our case the recent infarct was found in the area supplied by the circumflex branch of the left coronary artery. This was occluded; correlated with this there was diminution in amplitude of the QRS complexes of the electrocardiogram. Other branches were involved to be sure, but there were no areas of recent infarction in the regions supplied by these branches. There may be, then, based on Smith's experiments, on Herrick's (one), Smith's (one), and Wearn's (one) cases and on our own, a rough correlation between occlusion of the circumflex branch of the left coronary artery and decrease in amplitude of the QRS complexes. But in one of Smith's, Herrmann's and most of Wearn's cases, exact correspondences are not to be found. If, in the future, observers will make specific statements about the occurrence of pain, exertion as the exciting cause, fever, leucocytosis, friction rub, and of changes in T-waves, QRS complexes, and rhythm of the electrocardiograms, and about the specific vessels found at autopsy to be involved in the acute coronary accident, then sufficiently accurate material will accumulate upon

which it will be possible to decide whether a closer correlation can be made between lesion and sign than is possible at present. Under the circumstances, then, exact correlation between lesion and electrocardiographic sign is impossible. Whether further refinements of study, either of the site of injury or of better definition of alterations in the electrocardiograms, will permit accurate diagnosis cannot now be known.

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THE ACTION OF DIGITALIS IN COMPLETE HEART-BLOCK

Its Toxic Influence on the Idioventricular Rate and Rhythm*

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THE limitation of treatment with digitalis in patients with complete heart-block is by no means established. Although the administration of the drug has in certain instances been accompanied by beneficial effects,[1, 2, 3, 4] in others it has been directly associated with grave arrhythmic accidents at times unfortunately ending fatally.[5, 6] It is such serious outcomes that have probably impelled competent observers to state emphatically "that safety demands that digitalis be avoided in any case with bradycardia."[7] Such overcautiousness, however, may deprive a patient with complete heart-block of the only remedial measure for the relief of distressing symptoms when heart failure sets in.

The main discrepancy in views seems to center on whether the beneficial results of the drug are due to an increase in the muscular contraction of the heart or to an augmentation of the automatic ventricular rate. It is held by some that a quickening of the slow idioventricular rate is a therapeutic result to be desired,[8] and an increase in rate has even been suggested as a possible indicator of the therapeutic effects of the drug.[9] It is claimed by others that a clinical improvement in patients with complete heart-block and decompensation has been demonstrable *only* after an increase of the idioventricular rate.[10]

From a study of 8 patients with complete heart-block and signs of heart failure who have been admitted to the wards of the Montefiore Hospital within the last two years and treated with tincture of digitalis, it has been concluded that the ventricular rate is increased appreciably only after the administration of large doses of digitalis; that is, doses that are greater than those commonly employed in therapeutics. Three of these cases are reported in detail, since each represents a distinct type of toxic digitalis action on the ventricles in complete heart-block. The increase in the ventricular rate following the administration of digitalis was accompanied in 2 patients by such distressing symptoms as breathlessness and fainting spells with visual disturbances and in 1 patient there were fleeting periods of total loss of consciousness, all of which passed away with the restoration of the dominant idioventricular rate and rhythm. In 2 of the patients, digitalis had the effect of initiating transient auricular fibrillation.

*From the Service of Dr. B. S. Oppenheimer, the Medical Division of the Montefiore Hospital.

REVIEW OF LITERATURE

It is well established from experimental observations on the mammalian heart that after the development of lasting auriculo-ventricular dissociation and complete heart-block following the destruction of the bundle of His, effective doses of digitalis may increase the slow rate of the idioventricular rhythm.[11, 12, 13, 14] It is possible to have a bigeminal rhythm or a rapid regular or irregular ventricular tachycardia of the independently beating ventricles depending upon the quantities of digitalis introduced into the circulation. In human beings, a notable increase of the dominant rate of the ventricles in complete heart-block in association with the administration of digitalis has not been observed except under unusual circumstances. In the majority of cases in which an attempt has been made to induce a change in the idioventricular rate by digitalis, no results could be obtained when therapeutic doses were used.[15, 16, 17] The few instances cited in the literature that are supposed to have shown positive changes deserve repetition.

Bachman[10] noted what he believed to have been a "conspicuous" increase in the ventricular rate, with a notable decrease in the auricular beat in a patient with complete heart-block following the administration of the tincture of strophanthus. His patient received 5 minims of the tincture three times a day for twelve days. Whereas before the administration of the drug, the ventricular rate averaged 23 beats per minute, a day later it had increased to 27 beats. On the sixth day of observation, after the dose had been doubled for the day, the ventricular rate of the patient was found to be 33 beats per minute. A careful review of Bachman's protocol reveals, however, that despite the persistent use of the drug in the doses mentioned, the ventricular rate was only 28 beats per minute at about the eighth day and remained at that level throughout the patient's period of observation. The total increase of the basic ventricular rate, therefore, did not amount to more than 5 beats per minute, which is, as will be later pointed out, a relatively normal variation in complete heart-block when the rate is counted over a prolonged period of time. Such small increments in the rate of the ventricles could be as well attributed to the variability of the inherent mechanism of the independently beating ventricles as to the action of the drug itself.

Meyer[6] recorded polygraphically a doubling of the ventricular rate in a patient with complete heart-block during the administration of the infusion of digitalis. In his patient the ventricular rate varied at first between 28 and 36 beats per minute and increased up to 70 beats per minute five days after medication was started. An analysis of his records, however, reveals that five days prior to the administration of the drug, the patient showed a totally irregular heart action with a ventricular rate of almost 80 beats per minute, which in the next few days gradually became more regular and decreased spontaneously

to an idioventricular rate that varied between 26 and 28. It is obviously impossible in so labile a rhythm as this case presents to attribute any definite acceleration of the automatically beating ventricles to the use of the infusion of digitalis. Besides, since there was no other adequate evidence of digitalization, and the patient died two days following the sudden increase in the ventricular rate, it is probable that the rise of the rate might have been an ante-mortem phenomenon, as has been observed in other patients with complete heart-block, independently of the use of any drug.[20]

Bastedo's case[5] is more convincing, although the increase in the ventricular rate of his patient was accompanied by the appearance of retrograde rhythm, a condition observed experimentally in the transition stages from partial to complete heart-block and usually considered as an index that the block is incomplete.[22] In his patient with heart-block, digitalis had the effect of bringing on short spells of doubling of the intrinsic rate of the ventricles with the onset of retrograde rhythm. In one of the polygraphic tracings from this case, the ventricular rate showed a jump from 26 to 54, the auricular rate dropped from 62 to 54, and the rhythm was reversed so that the auricular systole followed that of the ventricles instead of preceding it, both having the same rate at the end of each such paroxysm. There was a long pause of the ventricles lasting some seconds following this change. During such variations of rhythm, the patient experienced passing attacks of faintness or lightheadedness though lying flat in bed.

It should be mentioned here, that in all of the three cases cited above, the suggested increase in the rate of the ventricles as a result of digitalis administration has been deduced from polygraphic tracings. By such means it is difficult to ascertain whether the increase in the ventricular rate is due primarily to stimulation of the idioventricular pacemaker or to premature beats of the ventricles. In some such instances even electrocardiograms may be difficult to interpret.

CASE REPORTS

CASE 1.—B. W., a woman, aged eighty-two years, was admitted to the Montefiore Hospital on September 26, 1927, and died on April 21, 1928. She complained of having had intermittent attacks of pain in the right upper quadrant for the past fifty years. Within the last two years she had been suffering from shortness of breath, precordial pains upon the least exertion, insomnia, headaches, and weakness.

Ten years ago (February, 1918) the patient was informed that she had gall bladder disease with gallstones but because of her age it was not thought advisable to operate upon her. With the exception of the periods when she experienced these recurrent attacks from her "gallstones," the patient was able to attend to her house duties until June, 1922, when she entered St. Luke's Hospital, complaining of a new series of symptoms which she developed in rapid succession. These were precordial pains, shortness of breath, and headache with weakness. The precordial pains came on with the least exertion and were paroxysmal in character. They were

accompanied by periods of restlessness and a sudden increase in respirations. During the seizures she would turn pale and cold and would be unable to speak. In half a minute after their onset she would feel better again but very weak. She remained at St. Luke's Hospital at first for about five weeks and after a short stay at home, had to be readmitted, when, because of the increasing severity and duration of her attacks and precordial pain, it was thought advisable to keep her in the hospital for the next fifteen weeks. She returned home but remained in bed most of the time during the following eight months.

In September, 1927, two weeks prior to her admission to the Montefiore Hospital, she had a very severe attack of pain over the precordial region for which she was treated at Bellevue Hospital. On the day of her admission to the Montefiore Hospital she had had a similar attack at home, but this time it was preceded and accompanied by loss of consciousness, convulsions, and incontinence of urine. It is not known whether her pulse rate was very slow during this seizure. She never had a similar experience during her entire period of stay at the Montefiore Hospital.

Fig. 1.—Case 1. Ecg. No. 3617. Jan. 4, 1928. Complete heart-block. Ventricular rate 37.3. Auricular rate 99. The ventricular complexes are all of the supraventricular form. The T-waves are all positive.

Physical examination revealed an old woman sitting in bed and reclining on several pillows. She was slightly dyspneic, but mentally clear. There was marked cyanosis of her lips, hands, and feet. The pupils were equal and both reacted to light and in accommodation. There was slight distention of the superficial jugular veins. The apical impulse of the heart was in the fifth intercostal space to the left of the midclavicular line. The heart rate was slow but regular and averaged 37 beats per minute. In the interventricular silences two auricular sounds could be heard near the fourth intercostal space to the left of the sternum. The aortic second sound was markedly accentuated. The pulses were equal and regular and of very good force. There was no pulse deficit. The radial arteries were thickened and slightly beaded. The blood pressure was 232/94 mm. It was the same on both sides.

Over the lungs posteriorly there was flatness, with absent breath sounds more marked on the right side than on the left. Many moist râles were heard over the extreme left base posteriorly. The abdomen was soft, lax, and there was a large

fluctuating and tender mass in the right upper quadrant. The liver edge was not palpable. There was no evidence of ascites. The lower extremities showed no edema.

Roentgen examination of the chest failed to reveal any abnormality of the lungs. The heart shadow showed slight enlargement of the left ventricle. The arch of the aorta showed a few calcific plaques.

The Wassermann reaction was negative on several occasions. The blood sugar was 106 mg. per 100 c.c. Urea nitrogen was 24.4 mg. per 100 c.c. The urine had a specific gravity of 1023, and there was no sugar or albumin; microscopic examination revealed a few pus cells.

Several electrocardiograms taken shortly after admission revealed the presence of complete auriculo-ventricular dissociation with complete heart-block, the ventricular rate averaging 37.3 beats per minute and the auricular rate being 99. There was

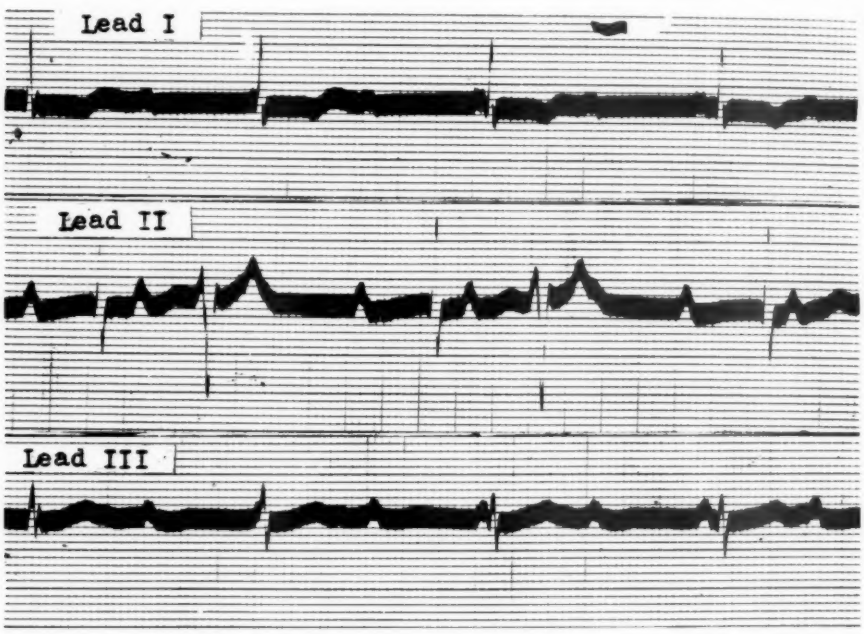

Fig. 2.—Case 1. Ecg. No. 3591. April 17, 1928, 10 A. M. Complete heart-block. Basic ventricular rate 48.4. Auricular rate 100. Lead II shows alternate premature beats of ventricle. T_1 is negative.

slight left axis deviation in some of the records. The ventricular complexes were all of the supraventricular form and the T-waves were all positive.

Course and Progress.—During her stay in the hospital the patient was able to be in a wheel chair for some of the time until March 14, 1928, when it was found necessary to perform thoracentesis of the chest which yielded 1500 c.c. of clear straw-colored fluid. From that time on, she was kept in bed, and an attempt was made to see if digitalis in adequate amounts would increase her idioventricular rate.

On March 27, 1928, the patient received orally 13 c.c. of the tincture of digitalis within twelve hours. (This was approximately a body-weight dose figured out by the Eggleston method.) She experienced no nausea or vomiting. An electrocardiogram taken on the following day revealed a slowing of the auricular rate from 99 to 78 beats per minute, but the ventricular rate remained practically unaffected,

showing an average of 35.3 beats per minute. There was definite evidence of digitalization in the electrocardiogram. The T-wave was negative in Lead I and reduced in size in both Leads II and III. P was now negative in Lead III. There were no significant changes noted in any of her electrocardiograms studied within the next few weeks, up to those taken on April 16, 1928, when the auricular rate showed a return to the original rate of 99 beats per minute and the T-waves had resumed their normal shape and size in every lead. (Fig. 1.)

On the morning of April 16, 1928, the patient complained of pain in the right side of her chest posteriorly near the angle of the scapula. Physical examination at this time revealed signs of dullness at the right base posteriorly, bronchovesicular

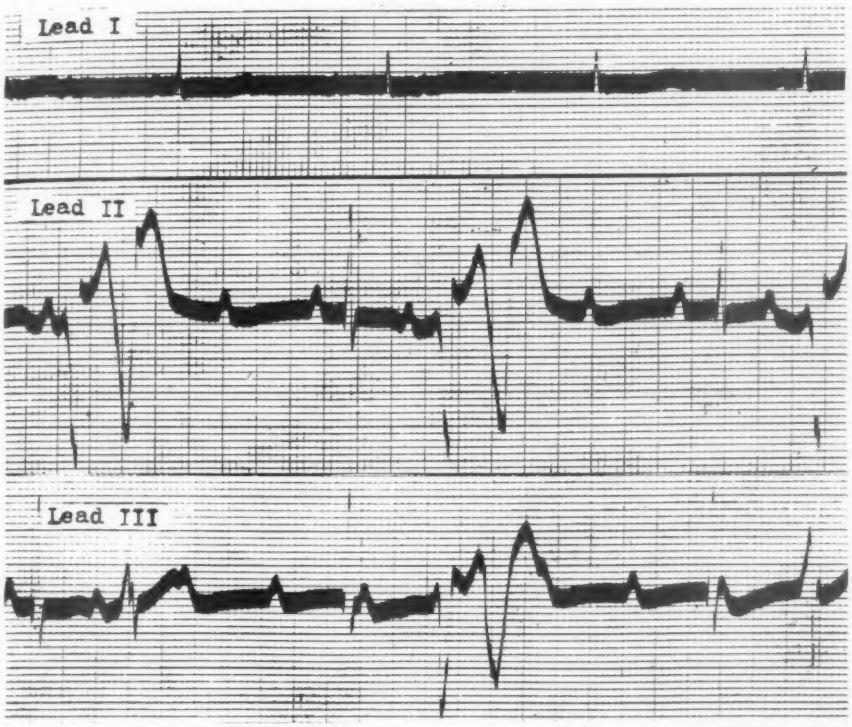

Fig. 3.—Case 1. Ecg. No. 3592. April 17, 1928, 5 P. M. Complete heart-block. Ventricular rate 48.4, Auricular rate 108.6. Bigeminal and trigeminal rhythm due to premature beats of ventricle.

breathing, and numerous moist râles. Because of the increase in respirations to 25 and the rise in temperature to 101.5° F., together with the physical signs, a diagnosis of bronchopneumonia was made. The ventricular rate, on this day, was never found to average more than 37 beats per minute when counted over long periods of time. At 11:50 A.M. of this day, the patient was given orally 6 c.c. of the tincture of digitalis. She received two more doses of the drug of 6 c.c. and 8 c.c. respectively within the next twelve hours, so that she received a total of 20 c.c. of the drug in a comparatively short time. Her condition was followed very closely and hourly heart rate and pulse readings were taken for the next seventy-two hours. These were checked by repeated electrocardiograms taken at various intervals during day and night. Despite the large doses of the drug, the patient

did not experience any nausea or vomiting. A definite increase of her ventricular rate independent of any premature beats was not noted until the following day.

April 17, 1928.—The general condition of the patient on this day was very poor. She was stuporous and in a semicomatose condition. The temperature was 102.3° F., the respirations were 26, and the pulse was intermittently regular and irregular but remarkably forceful. The signs of bronchopneumonia had spread to the opposite lung.

The electrocardiograms taken on the morning of this day showed a definite increase in the idioventricular rate which varied between 47.6 and 48.4, not counting the premature beats. The auricles now averaged 100 beats per minute. At times the heart rate would be regular for very long periods. At other times there were alternate premature beats of the ventricles, causing a bigeminal, trigeminal, and frequently quadrigeminal rhythm. Sometimes during the bigeminal periods, the alternate premature beats of the bigeminy would first assume the character of a

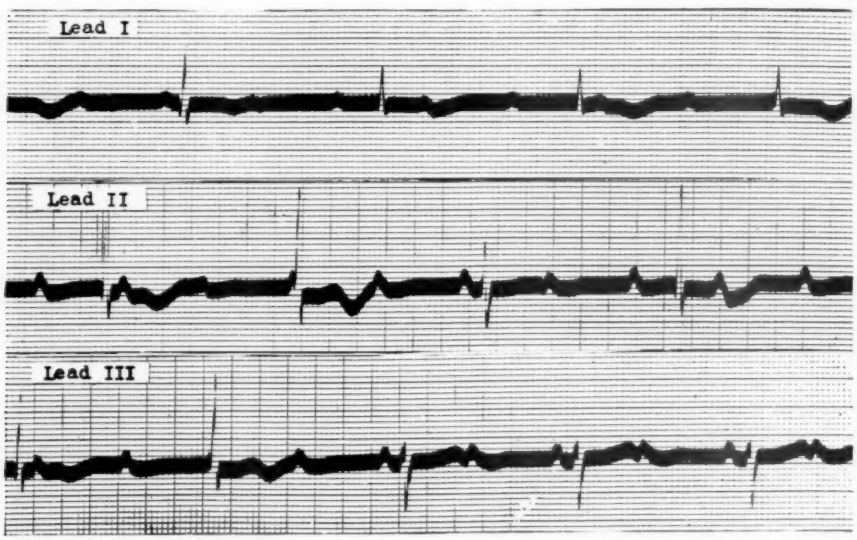

Fig. 4.—Case 1. Ecg. No. 3596. April 18, 1928, 11 A. M. Complete heart-block. The ventricular rate varies from lead to lead. It is 35.7 in Lead I, 43.4 in Lead II, and 42.8 in Lead III. Lead III shows an abrupt change from complete to partial heart-block which is accompanied by a transition from a dextrocardiogram to a levocardiogram. Note that the QRS group as well as the T-waves is affected by this change. The T-waves are now negative in all the leads showing complete heart-block.

dextrocardiogram and then suddenly change to a levocardiogram. The returning cycle following the premature beats of the bigeminy was always equal to the cycle of the idioventricular beat immediately preceding when the latter was not accompanied by an extrasystole. There were no changes in the size, shape, or form of the ventricular deflections at this time (Fig. 2).

Electrocardiograms taken between 2 and 5 P.M. of this day revealed no appreciable change in the ventricular rate from that noted above. The auricular beats, however, showed an increase to an average of 108.6 beats per minute. There were now to be noted definite variations in the initial ventricular deflections. These consisted in marked lowering of the voltage of the QRS group in Lead I and similar but slighter changes in Lead II. The T-wave was distinctly smaller in all three leads, with a tendency to negativity in the first lead. The P-wave was definitely increased in size. The predominating irregularity consisted of a bigeminal and

trigeminal rhythm due to premature ventricular beats some of which showed an abnormally wide deflection. At times there were long intervals when the ventricular rate was regular (Fig. 3).

April 18, 1928: On the morning of this day the patient appeared slightly better in that she was able to take some nourishment. The pulse rate was regular and very forceful. The average ventricular rate, counted over long periods of time, was 48 beats per minute. The electrocardiograms confirmed the absence of any irregularities until late in the day. Of particular interest in the records of this afternoon were the sudden and abrupt alterations in the form of the ventricular

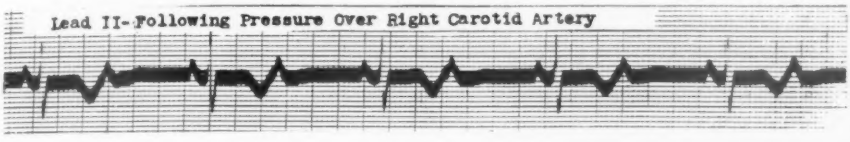

Fig. 5.—Case 1. Ecg. No. 3597. April 18, 1928. Lead II only. Partial heart-block, 2:1, following pressure over the right carotid artery. Note the slowing of both the auricular and the ventricular rates. The auricular rate has been reduced by this method from 100 beats per minute to 90.9 and the ventricular rate is now 45.4, whereas it was 48.2 before. Note that the T-wave is markedly negative now as compared with the single atypical complex of Lead II in Fig. 4.

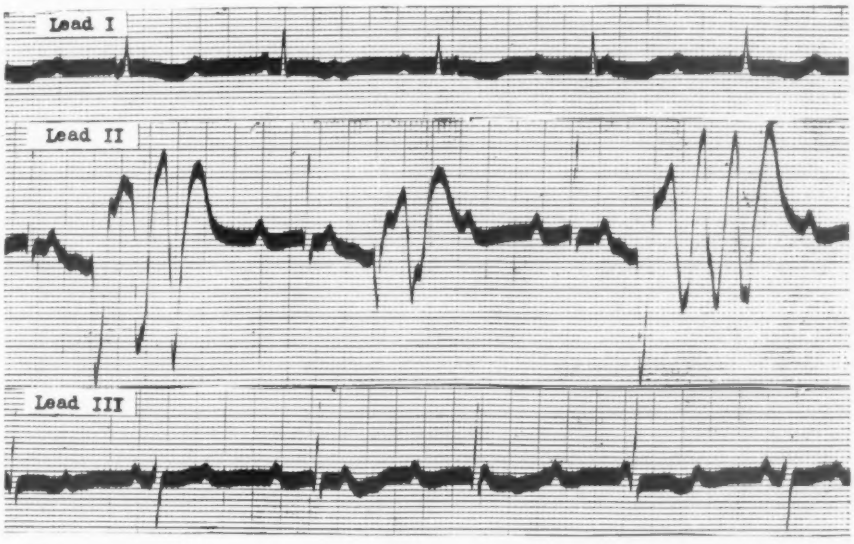

Fig. 6.—Case 1. Ecg. No. 3602. April 19, 1928. Complete heart-block. Basic ventricular rate is 53.5. Auricular rate is 125. Lead II shows premature beats of the ventricles arising from multiple foci, bringing the rate of the ventricles at such times as high as 300 beats per minute.

complexes of the electrocardiograms, which included the QRS groups as well as the T-waves. The QRS changed suddenly from a dextrocardiogram to a levocardiogram and the T-waves assumed a positive direction from a formerly negative one. When this took place there was a concomittant change from what was formerly a complete heart-block to what appears as a partial and incomplete 2:1 heart-block, with a sudden increase of the dominant idioventricular rate from 42.8 beats per minute to 48.2 beats per minute. When these abrupt variations in rate appeared in the electrocardiograms, the P-R interval always measured about 0.180 sec. They were present in all three leads, and it was particularly noticeable that the QRS as well

as the T portion of the ventricular complexes showed changes at the same time (Fig. 4). (It is to be noted that in some of the records of the previous day the change involved only the QRS complex, the T-wave remaining the same.)

Pressure over the left carotid sheath, at a time when the ventricular complexes did not show any of the variations described above, resulted in what appeared to be a change from a complete heart-block to that of partial 2:1 block with a decrease in the ventricular rate from 48.2 to 45.4 beats per minute and a slowing of the auricular rate from 99.3 to 90.9 beats per minute (Fig. 5).

April 19, 1928: The patient was now in coma, but the respirations were regular and 30 per minute. The temperature was 103.2° F., and the signs previously noted at the periphery of the lungs had now spread to the central part. The pulse was very forceful and again, as on the previous day, there were intermittent periods of regular and irregular rhythm. The ventricular rate of the dominant idioventricular rhythm was now 53.5 beats per minute and the auricular beats averaged 125. It was difficult at times to make out the arrhythmia of the heart clinically,

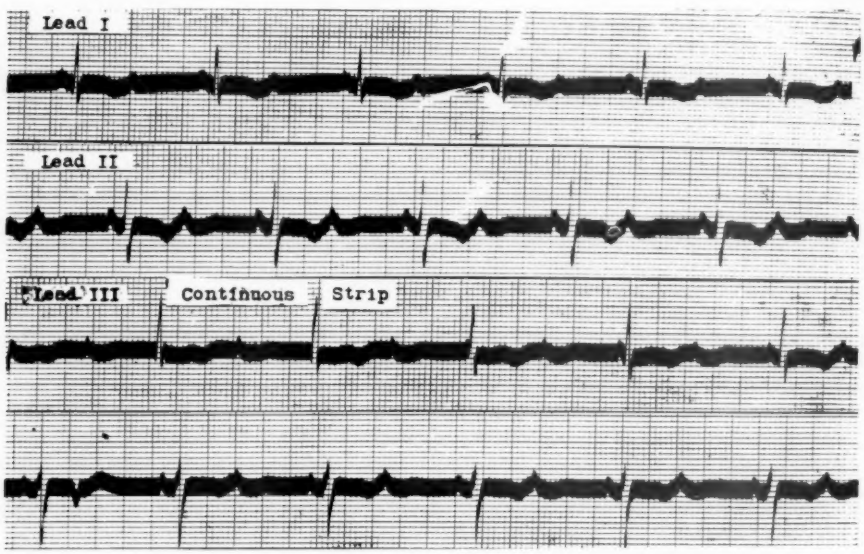

Fig. 7.—Case 1. Ecg. 3604. April 20, 1928, 1 A. M. Partial heart-block, 2:1. Lead III shows transition from complete to partial heart-block. Ventricular rate is 54.8, the auricular rate is 115.3. Note the change in rate with transition of complete heart-block to transient heart-block. The rhythm is now regular.

but the electrocardiograms revealed frequently innumerable premature beats of the ventricles with complexes of extremely wide deflections following the normal beats in groups of from 3 to 5 beats at a time. The impulses appear to be arising from several foci at the same time and are superimposed upon each other. Their presence brings the rate of the ventricles as high as 250 beats per minute. Changes in the ventricular deflection are still present, and they appear as abruptly as in other records but are especially noticeable in Lead III. Again it is to be noted that a sudden transition from a dextrocardiogram to a levocardiogram invariably takes place only at a time when the P-R interval measures a certain length, now approximately 0.160 sec. or 0.180 sec. In these records both the QRS and the T-waves are changed at the same time, that is, when the QRS complex assumes the form of a levocardiogram, the T-wave becomes positive (Fig. 6).

April 20, 1928: The patient was distinctly worse this morning. She was now totally unconscious and incontinent of feces and urine. The temperature was

103.4° F., the respirations were 30, and the pulse rate was 60 and almost perfectly regular. There were no premature beats to be heard at all. The interesting feature of the electrocardiograms on this day was the alternate periods of intermittent partial and complete heart-block. The transition from complete heart-block to partial heart-block (2:1) was associated with an abrupt change of the initial ventricular deflections from a dextrocardiogram to a levocardiogram and increase in the ventricular rate from 53.5 to 60 beats per minute. There were no coincident changes appreciable in the auricular beats which now averaged 93.7 beats per minute (Fig. 7).

April 21, 1928: On the morning of this day pulmonary edema set in. The temperature and respirations were about the same as on the previous day. The pulse was very forceful and regular. The ventricular rate averaged 65.2 beats per minute, and the auricular rate was now at its highest level of 125 beats (Fig. 8). The electrocardiograms showed complete auriculoventricular dissociation with complete heart-block. The T-waves were negative in all three leads. The highest rate recorded was 71 beats per minute, when the ventricular rhythm was known

Fig. 8.—Case 1. Ecg. No. 3607. April 21, 1928, 10 A. M. Complete heart-block. Ventricular rate is 65.2. Auricular rate is 125. The rhythm is regular. The T-waves are all markedly negative, showing the patient to be deeply under the influence of digitalis. Compare with Fig. 1.

to be regular. The rate kept up at about that level until 6 P.M. of this day when, because of the patient's general condition, observations had to be discontinued. She died on the evening of this day.

Comment.—In this patient a therapeutic dose of digitalis administered within twelve hours produced a slowing of the auricular rate from 99 beats to 78 beats per minute but did not accelerate the ventricles. Three weeks later, after the effects of the drug had worn off as judged by the return to normal of the auricular rate and the T-waves of the electrocardiogram, the patient was given orally approximately one and a half times the first dose of digitalis within twelve hours. One day following the administration of this amount of the drug, the dominant ventricular rate showed a sudden increase from 37.3 to 48.4 beats per minute. From then on the increase in the idioventricular rate was progressive until it reached 65.2 beats per

minute one day prior to death, independently of the presence of any premature beats. The auricular rate showed a similar progressive increase until it averaged 125 beats per minute, the highest figures recorded in the electrocardiograms.

The possibility must be considered that the bronchopneumonia was responsible for the increase in the ventricular rate. While it is well known that fever increases the heart rate of patients with normal sinus rhythm, in complete heart-block observations on this point are lacking. In one patient with lobar pneumonia who did not receive any digitalis, who was followed at the Montefiore Hospital, the rise in temperature due to the lung infection did not influence the rate of the ventricles. It would seem probable that in complete heart-block due to organic involvement of the bundle of His, the factor of pyrexia may be discounted as responsible for increasing the ventricular rate.

Again it may be questioned whether a uniform rise in the dominant ventricular rate might not have been an ante-mortem acceleration as has been observed by De Graaf and Wise in one of their patients with complete heart-block. The presence of simultaneous irregularities in rhythm in the electrocardiograms, such as multiple premature beats and the inversion of the T-wave, which became more prominent in all three leads from day to day, lend weight to the view, however, that both the increase in rate and the disturbances in rhythm are due to one and the same factor, namely, excessive digitalis administration.

CASE 2.—M. L. L., aged sixty-nine years, a peddler, was first seen on November 4, 1925. He had been in good health up to one year before (June, 1924) when he first began to complain of shortness of breath, progressive swelling of the lower extremities, and paroxysmal precordial pain, especially at night. He also suffered from pain over the right upper quadrant. Although at the beginning of his illness he could attend to his work, in the last six months he had been confined to bed because of weakness and swelling of the abdomen and scrotum.

Physical examination at this time (November 4, 1925) revealed an old, cyanotic man whose breathing was labored and of the asthmatic type, with prolonged wheezing expirations. There was cyanosis of the lips, cheeks, and finger nails. The superficial vessels of the neck were markedly distended and showed definite visible pulsations. The chest was moderately barrel-shaped.

The heart was markedly enlarged, the left lower border reaching the sixth intercostal space in the anterior axillary line. The heart sounds were of fair quality. A loud blowing systolic murmur replaced the entire first sound at the apex. The aortic second sound was accentuated. In the long diastolic silences, two and sometimes three muffled sounds could be heard best at the third intercostal space to the left of the sternum. These sounds corresponded with the systolic pulsations in the superficial veins of the neck, which were seen in the absence of a corresponding apical beat. The pulses were equal in force and time and were Corrigan in type. The rate was only 32 beats per minute. The radial arteries were tortuous, beaded, and rigid. The blood pressure varied between 300/80 and 210/80 mm. There was marked dullness over both lungs posteriorly. On the right side breathing was diminished from the angle of the scapula to the base. The abdomen was distended and there was shifting dullness. The liver was enlarged to 20 cm. below the costal margin. It was painful and tender to touch, but it was not pulsating. The

scrotum was markedly edematous and enlarged to the size of a grapefruit. The thighs and legs were extremely swollen. The prostate was large and nodular.

Course and Progress.—On daily doses of 3 c.c. of the tincture of digitalis orally for a period of twenty days, the edema of the scrotum and extremities cleared up completely, the ascites disappeared, but the fluid in the chest had to be removed by thoracentesis. In three months' time his weight came down from 194 pounds to 136. He was able to be up and about and no longer had to be restricted in his fluids. The liver, however, did not recede and remained practically the same throughout the rest of his life.

On April 14, 1926, the patient began to show symptoms of prostatic obstruction. He could not void, and on several occasions had to be catheterized. Shortly after the onset of these symptoms, he again began to experience shortness of breath. In two days' time, there was definite evidence of fluid in the right chest and swelling of the legs. An electrocardiogram taken at this time revealed the presence of complete heart-block with a ventricular rate of 88. Because of the increasing symptoms of heart failure, the patient was admitted to the wards of the Montefiore Hospital.

On April 23, 1926, he was given orally 10 c.c. of the tincture of digitalis, and

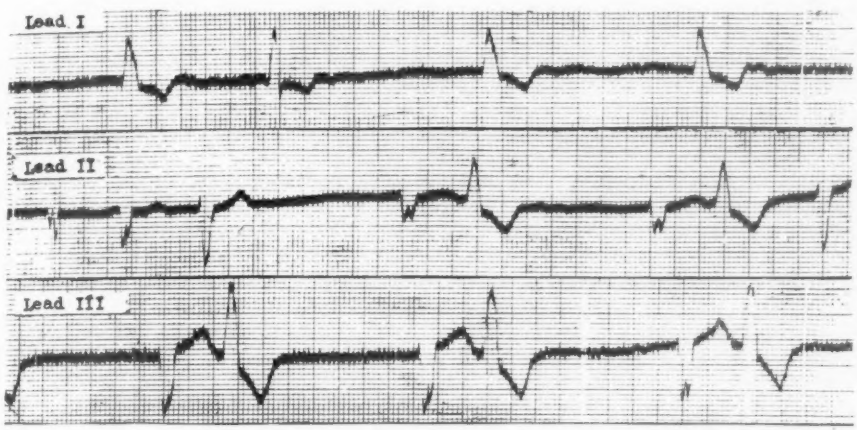

Fig. 9.—Case 2. Ecg. 1837. May 3, 1926. Complete heart-block. Auricular fibrillation. Premature beats of ventricle singly and in groups. In Lead II they are interpolated at both ends of the record.

this was followed within the subsequent three days by 10 c.c. more, so that he received a total of 20 c.c. of digitalis in three days. Two days after he received his last dose there was very marked increase in the heart rate, which became slightly irregular and averaged about 56 beats per minute. It was noted at the same time that the superficial jugular veins no longer showed the regular pulsations seen on previous occasions when his heart rate was regular. Clinically it was very difficult to distinguish the irregularity present. An electrocardiogram revealed complete heart-block, auricular fibrillation, and premature ventricular beats both isolated and in groups, many of which were interpolated (Fig. 9). At times these formed a bigeminal and trigeminal rhythm, but there was no definite sequence in their appearance. With the onset of these abnormal rhythms, the patient experienced fainting sensations and periods of momentary loss of consciousness. He complained that every other minute everything got "black" before his eyes.

All medication was discontinued except the usual sedatives. One week later all ventricular premature beats had disappeared, but the auricular fibrillation persisted. It was not until a little over six weeks after the onset of these arrhythmias that restoration to the normal basic rhythm of complete heart-block was observed. Clin-

ical examination at the time, June 6, 1926, revealed a regular ventricular rate of 32 beats per minute. In the long interventricular silences, there were two definite jugular pulsations accompanying two weak heart sounds now best heard at the level of the fourth intercostal space to the left of the sternum. The lungs were clear; the liver, however, remained enlarged as previously. There was no edema of the legs.

The patient remained in fairly good condition for several months after this episode, but at no other time was fibrillation of his auricles noted either clinically or graphically. He died suddenly while sitting in a chair on September 24 of this year.

Comment.—The dominant idioventricular rate of this patient was not influenced at all during the administration of daily doses of 3 c.c. of the tincture of digitalis over a period of twenty-one days, although the general condition and symptoms improved remarkably during that time. As a matter of fact careful measurements of the electrocardiogram revealed that at the time he received his first course of digitalis, from which he benefited considerably, the ventricular rate was even slower than at any subsequent observation. The ventricular rate showed an appreciable increase only after the administration of large doses of digitalis within a comparatively short time. The increase was due primarily to the interpolation of premature beats of the ventricle arising in multiple foci. The dominant rhythm was completely disrupted by these when they appeared in groups, since many of them showed no definite sequence in their appearance. That the auricular fibrillation and the multiple premature beats were due to excessive digitalis action is suggested by the fact that the withdrawal of the drug resulted in the restoration of the dominant rhythm of complete heart-block.

The increase of the ventricular rate due to premature beats as a result of digitalis was a very undesirable effect in this patient because there were momentary periods of loss of consciousness at a time when the ventricular rate was a high as 107 beats per minute. On the other hand, his symptoms were best relieved by moderate doses of digitalis, and he remained free from edema and breathlessness as long as the drug was used in this manner. His death was probably due to acute coronary vessel closure and was not related to the administration of the drug. There was no increase in the ventricular rate shortly before his death.

CASE 3.—L. R., man, aged fifty years, a tailor, was admitted to the Montefiore Hospital on August 5, 1927, and died on November 28, 1927. He had previously been observed at this hospital for a few days, leaving of his own accord to enter another institution.

The patient had been well and up and about until three years prior to admission (February, 1924), when he began to complain for the first time of difficulty in walking because of oppression in the epigastric region, which was worse on effort. Soon after, he became aware of breathlessness on exertion and noticed progressive swelling of his lower extremities during the daytime. Both of these symptoms

compelled him to seek medical attention in November, 1924, but it was not until one year later (November, 1925) that he entered a hospital. In the period of the next two years he was admitted to several institutions on nine separate occasions, besides being in constant touch with the out-patient clinics of four other hospitals from which we were able to obtain a consecutive story of his progress since the onset of his illness.

In the middle of the summer of 1925, the patient noticed that he could not walk a block in less than fifteen minutes because he had to stop every few steps to get his breath. At about the same time the swelling which was first limited to his feet progressed to his thighs and this increased his difficulty in getting around. Occasionally he would get nocturnal attacks of shortness of breath. In the next few months he observed that while at first the edema of the legs would disappear at night, toward the early part of November, 1925, the swelling persisted and was getting worse even though he remained in bed for the entire day. His first hospital admission was on November 10, 1925.

Physical examination at this time revealed an obese and well-preserved man who looked much younger than his age. He was sitting up in bed supported by several pillows, and breathing with difficulty. There was marked cyanosis of his lips and finger nails. The superficial veins of his neck were not distended. His chest was moderately barrel-shaped. The apical impulse of the heart was neither visible nor palpable and percussion of the cardiac outline was difficult because of the rounded contour of his chest. The heart sounds were of fair quality, the first sound at the apex being replaced by a soft blowing systolic murmur which could be heard all over the chest. The heart rate was 72 beats per minute, and there was an occasional premature beat with a compensatory pause. The pulses were of good quality. The radial vessels were slightly thickened and tortuous. The blood pressure was 144/90 mm. The lungs showed a moderate amount of moisture at both bases posteriorly. The abdomen was soft but large and lax. The liver edge could be felt 2 cm. below the costal margin. The spleen was not palpable. There was marked edema of the legs and thighs.

The Wassermann reaction was negative. The urea nitrogen was 21 mg. per 100 c.c. The urine did not show anything abnormal. An electrocardiogram taken at this time (November 11, 1925) revealed the presence of high grade intraventricular block. The QRS measured 0.160 sec. The P-R interval was prolonged to 0.280 sec. There was an occasional premature ventricular beat. Within the next few days there was a spontaneous increase of the P-R interval to 0.320 sec. with partial A-V block and dropped beats. Because of the presence of these signs, it was not deemed advisable at this time to give the patient any digitalis. His edema cleared up with rest in bed, restriction of fluids, and diuretics, so that he was able to leave the hospital in fairly good condition one month after his first admission. At the time of his discharge he was able to walk around very comfortably but slowly.

In the next few months, the patient attended an out-patient clinic but because of increasing discomfort from traveling to the clinic, he resorted to a local physician, who promptly placed him on graded doses of digitalis. (The dose is not known.) On his second admission to the hospital (September 24, 1926) the patient showed marked ascites and hydrothorax, both of which required immediate relief. He was very uncomfortable, his liver was now 5 cm. below the costal margin, and he showed marked edema of the legs. He was given no further medication except the usual sedatives for the night. Several electrocardiograms taken during the two months following his second admission revealed the presence of heart-block with a ventricular rate that varied between 40 and 44 beats per minute. At first the block was incomplete and showed a PR interval of 0.20 sec., with marked negativity of the T-wave in Leads I and II. It finally became complete and remained so until December, 1926, when there was a severe release of the block to an incomplete

one with 2:1 rhythm. The development of the complete heart-block was attributed to excessive digitalis therapy which the patient had received while at home. Nevertheless, between December 8, 1926, and January 13, 1927, the patient was again treated with digitalis because of the recurrent ascites and hydrothorax. In that period he received a total of 69.5 c.c. of the drug at the rate of 30 minims, at first only once a day and then at intervals several times a day. He did not develop complete heart-block again until January 28, 1927, but despite these arrhythmic variations he was feeling much better under the influence of the drug than without it.

Between this last date and his admission to the Montefiore Hospital he had to be admitted to the wards of several institutions because of dyspnea from excessive accumulation of fluid in his chest. From repeated electrocardiograms taken in this interval, it was noticed that his rhythm varied between a complete heart-block and an incomplete one depending upon whether or not he was under the influence of digitalis. As his illness progressed, it could definitely be seen, however, that he no

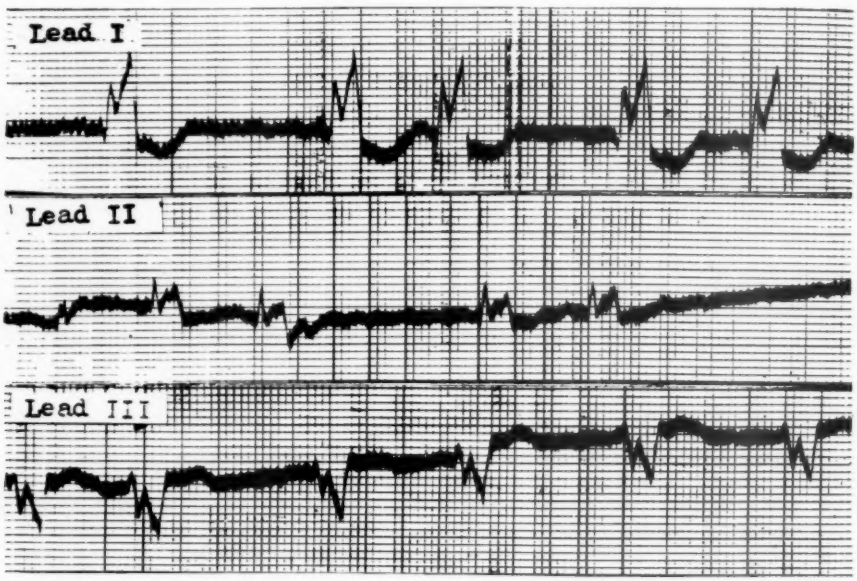

Fig. 10.—Case 3. Ecg. No. 2813. Aug. 9, 1927. Complete heart-block. Auricular fibrillation. Acceleration of the ventricular rate due to stimulation of the idioventricular pacemaker. Ventricular rate averages 87 beats per minute. Compare with following record.

longer responded to the drug as he had in the previous two years. Whereas, formerly his edema and breathlessness had become better with the use of digitalis, now, in the summer of 1927 he required more diuretics and abdominal taps for relief of symptoms. His ventricular rate during most of this time did not vary more than 8 beats per minute when he showed complete heart-block. Of unusual interest is the fact that premature beats were not observed in his latter records when it was well known that he was under the influence of digitalis.

In the ten days prior to his admission to the Montefiore Hospital he had received 2 c.c. of the tincture of digitalis once a day for the first three days and the same dose three times a day for the remaining seven days. Although when first seen in our wards, it was known that he had a complete heart-block with a ventricular rate of 40 and an auricular rate of 68 beats per minute, because of his very marked generalized anasarca, it was thought advisable to try further dig-

italis medication. His weight being 215 pounds, he was given 8 c.c. of the tincture of the drug on his admission. When seen two days later, he was so dyspneic that the only way in which he could find comfort was by sitting across the bed with his legs hanging over the side and his head bent over his chest. He was intensely cyanotic. His nose, lips, and ears were almost black. Because of the swelling of his face and neck, the superficial jugular veins were not visible. The apex impulse of the heart was neither visible nor palpable, and percussion of the cardiac outline was difficult because of the contour of his chest. The heart sounds were of poor quality, the first sound at the apex being replaced almost entirely by a loud systolic murmur which was heard best near the apical region. The heart rate was rapid and totally irregular. It varied from minute to minute and averaged 98 beats per minute when counted over a prolonged period of time. At times on auscultation there was definite bigeminal rhythm, and at other times there were groups of beats coming in pairs but irregularly spaced. At other times the irregularity had the rhythm of a definite quadrigeminy. The pulse was of good quality and forceful. There was no deficit. The lungs were remarkably free from moisture. The ab-

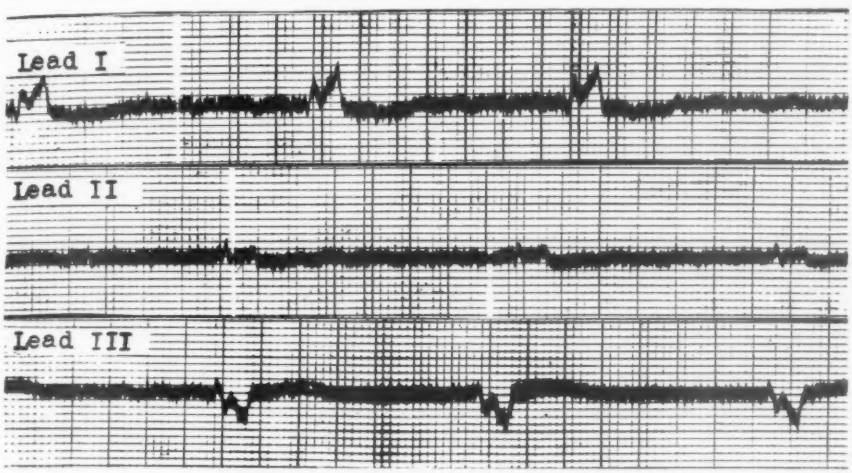

Fig. 11.—Case 3. Ecg. No. 2826. Aug. 24, 1927. Complete heart-block. Auricular fibrillation. Ventricular rate now averages 40 beats per minute. Note the disappearance of ventricular arrhythmia. The voltage of QRS is lower than in previous record.

domen was slightly swollen, and there was shifting dullness. The liver edge was down to the level of the umbilicus and the legs were extremely swollen.

An electrocardiogram taken at this time (Fig. 10) reveals a variable ventricular rate with a maximum of 93 beats per minute. The beats show a tendency to coupling. The QRS complexes are 0.20 sec. in duration and markedly notched. Their voltage is variable from lead to lead, the maximum being 12 mm. high in Lead I. There is no definite evidence of a P-wave to be seen in any of the leads. The ventricular complexes resemble one another very much from beat to beat so that it is impossible to state which is the dominant beat of the rhythm. Digitalis was discontinued, and the patient did not receive any more of this drug during the rest of his period of observation.

He was made comfortable with adequate opiates, and in the next few days he showed some improvement in that he was able to lie down comfortably and take nourishment. His chest and abdomen were tapped again within a week, but the edema of the legs persisted until the end. On August 24, 1927, the electrocardiogram showed very clearly a complete heart-block, with a ventricular rate varying between

38 and 42. The voltage of the QRS complexes was distinctly lower and there were no evidences of regular auricular activity which was now replaced by auricular fibrillation (Fig. 11).

On September 7, 1927 almost one month after his admission, the patient was looking and feeling slightly better. Although his legs were swollen, he was able to eat and sleep comfortably and even asked to be wheeled around in a chair for a while each day. On this day his rhythm was almost perfectly regular. His pulse rate was 40 and in the sitting position, with his head in slight dorsal extension, it was possible to see the jugular pulsations in the interventricular silences. The electrocardiogram (Fig. 12) confirmed the regularity of the heart-block and in addition revealed the presence of a regular auricular rate at an average of 88 beats per minute. The QRS complexes were distinctly lower than at any other previous examination.

The complete heart-block persisted with but very little variation in both the auricular and ventricular rate until he died from cardiac failure on November 27, 1927. On the day of his death, the ventricular rate fell to about 10 beats per minute shortly before he expired.

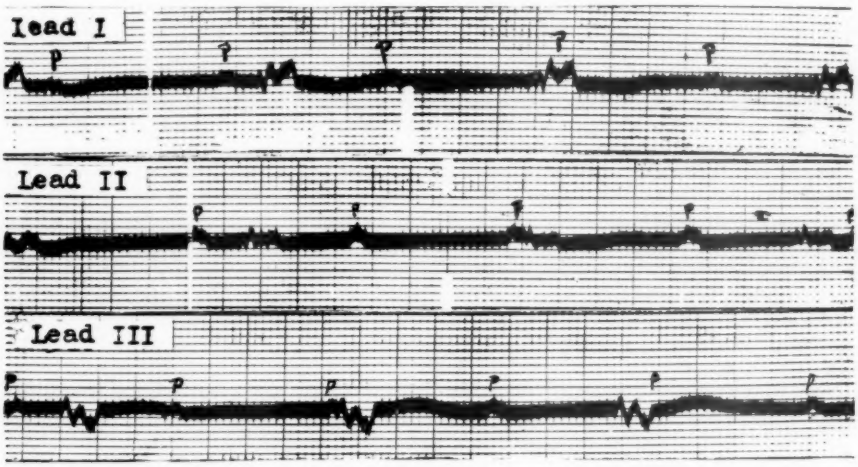

Fig. 12.—Case 3. Ecg. 2844. Sept. 7, 1927. Complete heart-block. Ventricular rate 40. Auricular rate 88. Note the auricules are regular now. Compare with previous record.

Comment.—This patient with marked myocardial involvement was followed over a period of two years. In the early stages of his illness he showed various degrees of block, but changes from one form of block to another were never accompanied by any Stokes-Adams seizures. A stage was reached in his illness when the block became permanent, and he no longer responded effectively to any form of medication. Despite the variable changes in rhythm that he exhibited from partial to complete heart-block, the patient benefited considerably from the administration of digitalis for over one and a half years after he began to show signs of heart failure with decompensation. At no time after the establishment of complete heart-block was there noted any increase in the ventricular rate except after he had received digitalis in excess of the usual doses to which he had been accustomed.

After the administration of 50 c.c. of the drug within eight days, he developed an irregular acceleration of the ventricles with auricular fibrillation. The ventricular irregularity disappeared within a few days, but the fibrillation persisted for several weeks.

DISCUSSION

An appreciation of the effects of any drug on the automatic beating ventricles in human beings must first take into account the inherent variability of the ventricles in complete heart-block as seen from day to day over a prolonged period of time. In summarizing such changes, Frey[19] has called particular attention to the fact that many patients with complete heart-block exhibit spontaneous premature beats in the slow interventricular periods. Usually there is no compensatory pause following these. Sometimes the premature beats are interpolated, but compensatory pauses may be present. Infrequently, a complete ventricular cycle may drop out with perfect regularity. The dominant beats of the ventricles may not be disturbed by such omissions. Furthermore, there may be periodic reductions and increases in the frequency of the idioventricular contractions so that they seem to resemble Luciani periods. These are seen particularly in patients subject to Stokes-Adams syndrome. For such reasons any study of the effects of digitalis on the rate of the ventricles in complete heart-block must be correlated with the antecedent rate and rhythm which the patient presents before the drug is used. Obviously if the patient's rate is very labile and varies from day to day, that amount of variability must be taken into consideration in judging the results produced by an effective dose of digitalis.

In the three patients reported in this paper, the dominant ventricular rate as counted over a prolonged period of several months during the presence of heart-block, never varied more than 10 beats per minute at any period. In all of the 3 patients spontaneous premature beats were recorded at some time during the course of their illness. In 1 case a single body-weight dose of digitalis caused the premature beats to disappear. In 2 patients the administration of a larger dose of digitalis than a body-weight measure resulted, however, in the production of numerous premature beats of the ventricle arising from multiple foci. In the first case all of these disappeared as the dominant ventricular rate increased from the toxic action of the drug. This is consistent with experimental findings where it is constantly observed that premature beats of the ventricle disappear when the ventricles themselves take on a higher rate than the inherent basic rate.

In this respect it is important to note how closely the appearance of the abnormal rate and rhythm in man may be compared with similar disturbances in animals, following the use of toxic doses of digitalis after the establishment of complete heart-block. For example, von

Egmond[14] has tabulated from his researches and from those of others a definite sequence of events following the absorption of digitalis in complete heart-block. In the first stage, there are no irregularities. The idioventricular rhythm remains the same and it is slow. A second stage follows which is characterized by the appearance of premature beats in a dominant ventricular rhythm of relatively low rate. The frequency of the ventricles may then increase suddenly to a very high rate which may be at first either regular or irregular, depending upon the amount of drug used.

The auricles may also beat fast, or if the vagus is influenced by the drug they may beat slowly, but this is characteristic of the first stage only. The slowing of the auricles is frequently followed at such times by a change from complete heart-block to what appears as a partial 2:1 heart-block or even a normal rhythm.[12] But in man the features of an incomplete heart-block with 2:1 rhythm have been considered hitherto only apparent and the consequence of a fortuitous change of the auricular to the ventricular rate under the slowing of digitalis on the auricles.[17]

In 1 of our patients, however (Case 1) the change from complete heart-block to partial heart-block is not apparent but real, since the P-R interval preceeding the QRS complexes in the records showing partial heart-block are equal from beat to beat. Besides in this patient during the stages of complete heart-block, pressure over the carotid artery in the neck resulted in no changes whatsoever in either the ventricular rate or rhythm, whereas during the appearance of the partial heart-block both the auricles and ventricles were slowed by carotid pressure. The explanation for this unusual phenomenon is not clear.

Nevertheless, an appreciation of these sudden changes in rate and rhythm during the administration of digitalis and the initiation of the abrupt forms of the ventricular tachycardias cannot be overestimated in man, for they may be the first index of overdigitalization in patients with complete heart-block who are receiving the drug.

A study of the electrocardiograms accompanying this report reveals that the increase in the ventricular rate in already established complete heart-block as a result of digitalis may be one of several types.

1. There may be a progressive uniform increase in the dominant rate of the ventricles independent of the presence of any premature beats.

2. A further increase in the rate after it has already been augmented by digitalis may take place by a sudden change from complete heart-block to partial heart-block. Such a transition may be seen in the records of Case 1, where the change in rate is also accompanied by a change in rhythm when a complete heart-block becomes a partial heart-block.

3. An increase of the ventricular rate may also occur from the stimulation of multiple ventricular foci, giving rise to premature beats. These may be single or in groups, forming a bigeminal, trigeminal, or quadrigeminal rhythm. In some instances the beats are interpolated while in others they may be superimposed on each other so as to bring the rate as high as 300 beats per minute at times. It is probable that ventricular fibrillation is the next step, although fortunately it has not been observed in our studies.

DIGITALIS AND THE DEVELOPMENT OF TRANSIENT AURICULAR FIBRILLATION IN COMPLETE HEART-BLOCK

Transient auricular fibrillation was observed as a toxic manifestation of digitalis administration in 2 patients with complete heart-block. In both patients the arrhythmia set in suddenly and without the electrocardiographic evidence, recognition would have been impossible. In one patient, the auricular irregularity was masked by the presence of premature beats, most of which were interpolated in groups (Case 2). These disappeared after three days, the polygeminal rhythm giving way to a trigeminal then to a bigeminal rhythm and finally to isolated premature ventricular beats and then to the dominant idioventricular rhythm. The sequence of events in the other patient was somewhat similar, but the ventricular irregularity was due to stimulation of the idioventricular pacemaker rather than to multiple premature beats of the ventricle (Case 3). The auricles continued to fibrillate, however, in both cases for approximately four and six weeks respectively. In the first patient, the intravenous administration of atropine sulphate, gr. 1/100, on the fourth day after the establishment of auricular fibrillation did not produce any changes in either rate or rhythm for one-half hour, during which time electrocardiograms were taken every five minutes. There were definite changes, however, in the initial deflections of the ventricular complexes at the end of the experiment, a phenomenon which had been observed in this patient prior to the administration of atropine.

The auricular rate was greater in both patients following the disappearance of the fibrillation than it was before its onset. For example, in Case 2, the rate was 66 before and 82 after, whereas in Case 3, the rate was 69.3 before and 88 after. It seems evident that in both cases digitalis was able to cause slowing of the auricular rate through vagal inhibition shortly before the appearance of auricular fibrillation.

That auricular fibrillation might be a toxic manifestation of digitalis in complete heart-block has been suspected by previous observers. In a patient with complete heart-block who had been receiving digitalis, Hewlett and Barringer[9] noted the disappearance of

the "a" wave from the jugular tracing and at first regarded the incident as a defect in technic, but the second disappearance from the apical tracing during a second course of the drug suggested to them that this may have been due to a "toxic weakening of auricular contractions." The presence of an extrasystole on each occasion also suggested to them a toxic digitalis effect. Similarly Neuhoff[20] studied graphically a patient with complete heart-block with a rhythmic ventricular rate of 35 and an auricular speed of 32 beats per minute, as demonstrated by polygraphic and electrocardiographic tracings. Through inadvertence on the part of the nurse, digitalis was administered for several weeks, after having been ordered discontinued. The pulse then became arrhythmic. The auricular beats were no longer heard in the interventricular silences and extrasystoles, most of which were frustrated, were audible at the apex. Regular recurrent auricular waves could not be identified in the jugular tracings. Two weeks after digitalis was discontinued, heart-block was again present as shown by the electrocardiogram, and the auricular sounds could be heard again. From the auscultatory evidence, from the absence of the auricular waves in the jugular tracing, and from the fact that digitalis poisoning sometimes induces auricular fibrillation and extrasystoles, it seemed probable to him that the polygraphic tracings represented heart-block, auricular fibrillation and ectopic beats, "a unique instance of digitalis poisoning in a patient with complete heart-block."

In complete heart-block, the sudden simultaneous appearance of auricular fibrillation and premature beats, whether singly or in groups, during the administration of digitalis may be considered sufficient evidence that the onset of the irregularities was initiated by the drug.[21] The abruptness with which these rhythms set in without any premonitory signs or symptoms, such as nausea and vomiting, or isolated premature beats, is by no means rare in certain patients with normal sinus rhythm.[22] There is no reason why it should not be seen in patients with complete heart-block. The important fact to remember is that the onset of auricular fibrillation is a sign of excessive digitalization or digitalis hypersusceptibility, and it is to be used as an indication for the withdrawal of the drug. Very recently the possibility has been suggested[23] that these patients really present auricular standstill, as it is observed experimentally in animals when too much of the drug is introduced rapidly intravenously. In human beings this is an exceedingly rare phenomenon, and even though the electrocardiograms show no evidence of regular auricular activity, there are other criteria that must be considered before making a diagnosis of complete standstill of the auricles.[24]

CONCLUSIONS

1. Digitalis in therapeutic doses may be of great benefit to patients with complete heart-block and heart failure. The improvement in these patients takes place through the direct action of digitalis on the heart muscle and not through an acceleration of the automatic beating ventricles.

2. In 3 out of 8 patients with complete heart-block studied at the Montefiore Hospital, the ventricular rate was increased appreciably only after the administration of digitalis in doses larger than those commonly employed in therapeutics.

3. The increase of the ventricular rate in complete heart-block during the administration of digitalis is a toxic manifestation of the drug, and because of the undesirable effects calls for its immediate withdrawal.

4. Transient auricular fibrillation is another manifestation of digitalis intoxication in complete heart-block.

5. Since it is difficult to appreciate the various types of toxic rhythms that manifest themselves in complete heart-block during digitalis therapy, it is advisable to prescribe the drug to such patients, under careful graphic control.

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THE INCIDENCE AND SIGNIFICANCE OF ELECTROCARDIOGRAMS SHOWING THE FEATURES OF LEFT AXIS DEVIATION AND QRS OF NORMAL DURATION WITH INVERTED T_1 AND UPRIGHT T_3*

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CLINICAL evaluation of heart disease centers more and more around the question of the integrity of the myocardium. In like manner the value of the electrocardiogram has come to depend chiefly upon the amount of information it gives on this question. In the application of electrocardiography to the diagnosis of heart disease, therefore, chief interest at present attaches to the form of the ventricular complex. Full understanding of all components of the ventricular complex, however, has not been reached, and as a consequence of this incompleteness, clinical interpretation of certain abnormalities is still somewhat equivocal.

The factors which cause the electrical axis to deviate to the right or to the left, and thus determine the direction of the QRS complex are fairly well understood and are applied clinically with a certain measure of exactness. But much uncertainty exists with regard to the T-wave. Clinical interpretation of changes in its form and direction, therefore, is still attended with considerable difficulty. Willius[1] and others have shown that inversion of the T-wave (except in Lead III) carries an unfavorable prognosis, and it has become customary to regard records which show such inversion as indicative of "myocarditis" (or drug effect). Little is known, however, of the exact pathological processes responsible for the inversion. Notwithstanding this uncertainty, or perhaps because of it, there has arisen a tendency to consider separately the T-wave and the QRS complex rather than to study both in their relationship to each other. It may well be questioned whether this separate consideration of the initial and final phases of the ventricular complex has not impeded progress in reaching an understanding of it, particularly so far as the T-wave is concerned.

As a consequence of this separate consideration of the components of the ventricular complex, and of the relatively more complete knowledge of the initial phase, it has been customary to diagnose records showing the QRS complex upright in Lead I and downwardly deflected in Lead III, with QRS of normal duration, as "left ventricular predominance" or "left ventricular preponderance," no matter what may

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be the direction of the T-waves in either the first or the third leads of such electrocardiograms.

Various combinations of the initial (QRS complex) and final (T-wave) phases of the ventricular complex in such records are shown in Fig. 1. Each record in this figure indicates deviation of the electrical axis to the left. The fourth is an example of the type with which our paper deals. Other examples of this type are shown in Fig. 2.

There is much evidence that electrocardiograms of this particular type result from defective conductivity in the right limb of the A-V bundle rather than from other causes of left axis deviation. This evidence rests on both experimental and clinical data. Wilson and Herrmann,[2] by producing levogram and dextrogram independently of each other, obtained various records showing the results of superimposing one on the other with different time relationships. When levogram and dextrogram were superimposed synchronously, the resulting rec-

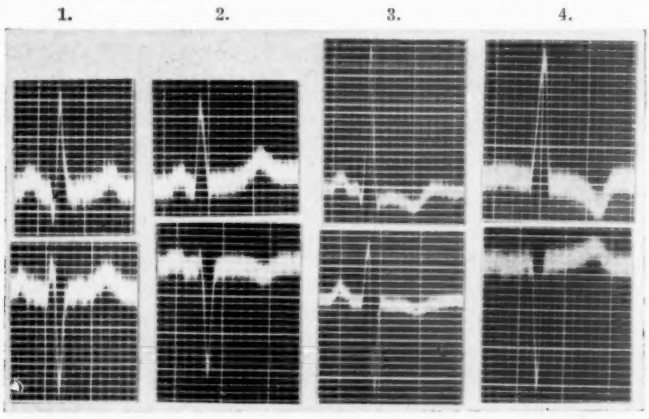

Fig. 1.—Electrocardiograms showing left axis deviation. In No. 4 the relationship of the T-wave to the QRS complex is that of defective conductivity in the right bundle-branch. In all figures Leads I and III are shown.

ord, of course, had the form of the normal bicardiogram. But when the levogram was caused to anticipate the dextrogram, the resulting bicardiogram exhibited not a haphazard relationship of T-wave to QRS complex, but showed the precise relationship exhibited in the records which constitute the theme of our paper. Under the conditions of the experiment there was relative delay of conduction to the right side of the heart.

Clinical data also support the conception that these records represent defective conductivity in the right branch of the bundle. Such evidence is presented in Fig. 3. This shows four electrocardiograms of the same patient. They were made on August 14, August 19, August 24, and September 8, 1923, respectively. The first and second records in this figure show right bundle-branch block. The third and fourth conform to the type under discussion. In view of the recent complete

block in the right limb of the A-V bundle, it would appear more reasonable to interpret the later records in this case as representing defective conductivity in the right limb of the bundle than to ascribe their shape to some other cause of left axis deviation. Such an interpreta-

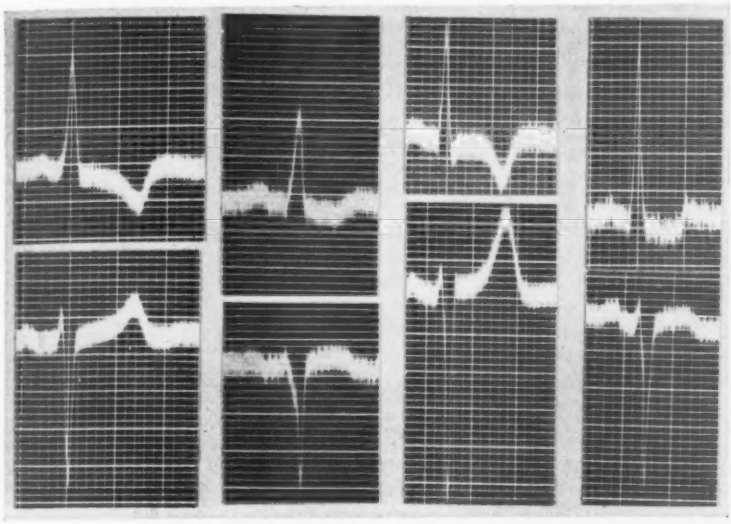

Fig. 2.—Additional electrocardiograms of the same type as No. 4 in Fig. 1.

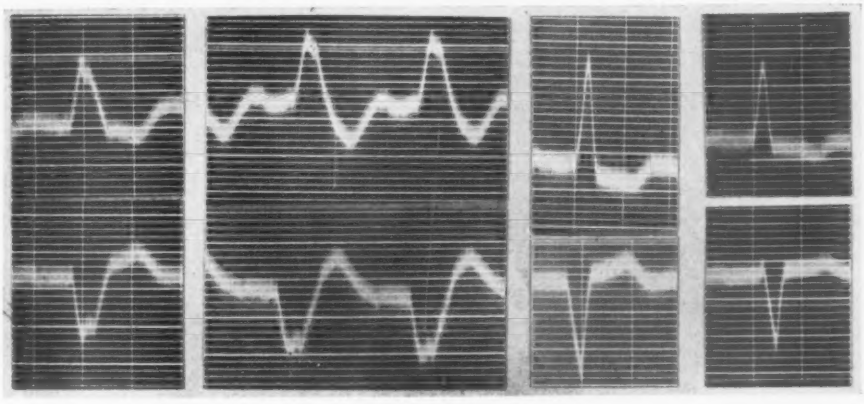

Fig. 3.—Four electrocardiograms from the same patient. The first was made August 14, 1923; the second, August 19, 1923; the third, August 24, 1923, and the fourth, September 8, 1923. The first two are indicative of right bundle-branch block. Note that the relationship of the T-wave to the QRS complex is the same in all four records.

tion is the only one which *alone* accounts for both the left axis deviation and the direction of the T-waves.

If impaired conductivity in the right branch of the A-V bundle, rather than relative left ventricular hypertrophy or other cause of left axis deviation, is the correct interpretation of the third and fourth

records in Fig. 3, the same interpretation may well apply to other such electrocardiograms obtained in cases in which right bundle-branch block has not previously been demonstrated. Records of this type and those showing right bundle-branch block may be expressions of the same process, which differs only in degree in the two instances. If so, the progress of heart disease in such a case might, on occasion, be followed electrocardiographically from the stage of delayed conduction in the right branch of the bundle to the stage of complete block in this branch. Electrocardiograms which give such a record of the progress of heart disease in a patient are shown in Fig. 4.

The patient in this case, a man fifty-four years old, had had hypertension for years. At the time of the first record, May 3, 1927, he had had few symptoms directly referable to his heart. In August, 1927, he

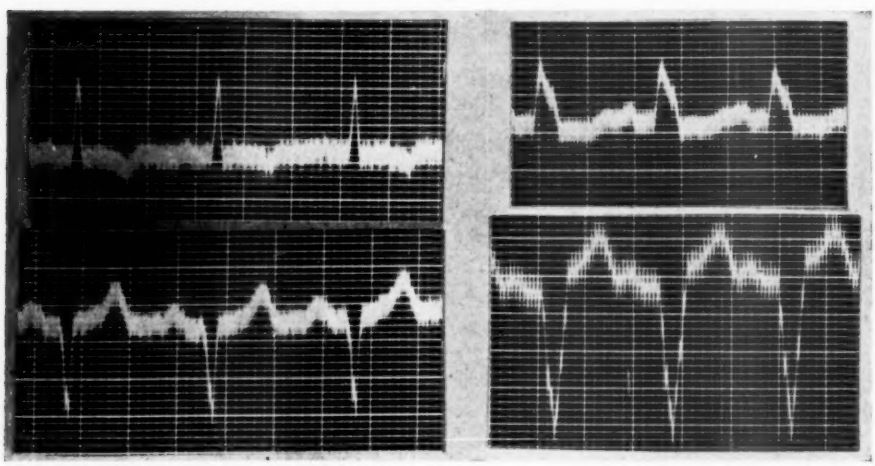

Fig. 4.—Two electrocardiograms from the same patient. The first record, made May 3, 1927, indicates defective conduction in the right bundle-branch. The second record, made October 25, 1927, shows right bundle-branch block. (Extraneous alternating current somewhat distorts the second record.)

had dyspnea, edema, and enlargement of the liver. The next record, October 25, 1927, shows right bundle-branch block. Subsequent records showed that the block persisted. This case offers additional clinical evidence that records conforming to the type described in the title represent impairment of conductivity in the right limb of the A-V bundle. It lends weight also to the suggestion just advanced that records of this type and those showing right bundle-branch block represent only different degrees of the same pathological process, and have, therefore, the same significance from the standpoint of etiological diagnosis.

The question whether these electrocardiograms do in fact represent defective right bundle-branch conduction is, of necessity, closely related to the question of etiology. What is the pathological process in

the bundle that is responsible for the impairment of conductivity? Is it the same in all, or at any rate in most, cases? An investigation of the significance of electrocardiograms of this type, therefore, involves two phases. First, an analysis should be made of all cases in which they occur in order to determine whether the cases present certain features in common. The results obtained from this study of incidence should then be applied to the question of impairment of conductivity. If certain features are indeed found to be constant, these should be investigated to see whether they might imply a conduction defect in the right limb of the A-V bundle.

It appeared to one of us that the incidence of these electrocardiograms is confined almost exclusively to cases of arteriosclerotic heart disease and hypertensive heart disease.* The primary object of our paper is to test the accuracy of that impression; to determine in fact, whatever their explanation, whether any common finding attends their incidence. The second object has been to apply the data thus acquired to the problem of defective conduction; to determine whether such data might indicate a pathological process of such character as to impair conductivity in the right branch of the A-V bundle.

METHOD OF STUDY

All electrocardiograms of the type described were withdrawn from our files. The criteria adhered to in the selection of records were the following: QRS deflection upright in Lead I, downwardly directed in Lead III; T-wave inverted or diphasic in Lead I, upright in Lead III; duration of QRS complex less than 0.10 second. The corresponding case histories were obtained and the following data noted: Age; sex; Wassermann reaction; highest systolic blood pressure; lowest diastolic blood pressure; all diagnoses both primary and secondary; whether or not there was a diagnosis of any cardiac abnormality; type of heart disease;† evidence of any degree of heart failure (past or present); evidence of cardiac enlargement; evidence of arterial sclerosis and condition of coronary arteries at autopsy.

RESULTS

There were 237 cases. The results are shown in Tables I, II and III. In 128 cases the electrocardiogram showed the T-wave completely inverted in Lead I, while in the remaining 109 cases the T-wave was more or less diphasic in the first lead.

*Such an incidence, if found to be correct, would at once associate them closely with records showing right bundle-branch block, and would lend still further evidence, therefore, to the conception that both result from the same pathological process.

†In the case of those histories which ante-dated the adoption of etiological classification in our files, the appropriate etiological diagnosis was supplied as well as possible from the data in the case history.

There were 150 males and 87 females. The age table shows the number of each sex that fall in each decade. The other tables are not subdivided on the basis of sex.

TABLE I

Showing the grouping of cases according to age and according to blood pressure.

AGE				BLOOD PRESSURE			
	NUMBER OF CASES			HIGHEST	NUMBER	LOWEST	NUMBER
YEARS	MALE	FEMALE	BOTH	SYSTOLIC	OF CASES	DIASTOLIC	OF CASES
Above 69	16	10	26	Above 249	22	Above 149	9
60-69	34	13	47	200-249	85	125-149	38
50-59	51	29	80	170-199	54	100-124	63
40-49	36	18	54	160-169	15	90- 99	31
30-39	10	13	23	150-159	16	80- 89	27
20-39	2	1	3	140-149	11	70- 79	18
Under 20	1	0	1	130-139	11	Under 70	39
No data	0	3	3	Under 130	14	No data	12
Total	150	87	237	No data	9		
				Total	237	Total	237

TABLE II

Showing the number of cases with and without: (a) arterial sclerosis, (b) cardiac diagnosis as principal diagnosis, (c) cardiac diagnosis as either principal or secondary diagnosis, (d) evidence of any degree of cardiac insufficiency, (e) any evidence of hypertrophy.

	ARTERIAL SCLEROSIS	WAS PRINCIPAL DIAGNOSIS A "CARDIAC DIAGNOSIS"?	DID DIAGNOSES (PRINCIPAL AND SECONDARY) INCLUDE A "CARDIAC DIAGNOSIS"?	WAS THERE EVIDENCE (PAST OR PRESENT) OF CARDIAC INSUFFICIENCY?	WAS THERE ANY EVIDENCE OF HYPERTROPHY?
Yes	163	97	132	131	207
No	47	137	102	100	23
No data	27	3	3	6	7

TABLE III

DIAGNOSIS 234

Showing division of cases into "Cardiac" and "No cardiac" groups, according to diagnoses on case history. The cardiac group is subdivided according to the type of heart disease. The non-cardiac group is subdivided according to whether or not a diagnosis of arterial sclerosis or hypertension appeared on the case history. Diagnoses in both groups above the dotted line carry more or less suspicion of coronary disease.

A CARDIAC 132			B NO CARDIAC 102	
Arteriosclerotic Hypertensive Angina	100	125	With: hypertension arterial sclerosis	96 (100)
Syphilitic Aneurysm	25			
Rheumatic	5	7	Without: hypertension arterial sclerosis	6 (2)
Bac. endocarditis	1			
Thyroid	1			

COMMENT

Age.—It will be observed at once that these electrocardiograms were found in people of middle age and beyond. Only 4 of the 234, whose ages were recorded, were under thirty years of age. Only 11 were less than thirty-five.

Blood Pressure.—These patients had hypertension. In 89 per cent of cases the systolic blood pressure at some time was recorded at least as high as 139 mm. In 70 per cent a reading at least as high as 170 mm. was recorded, while in 46.9 per cent a reading of 200 mm. or higher was found. The diastolic blood pressure was never recorded below 100 in 48 per cent of the cases. It was never below 80 in 74 per cent. The relatively large number with a diastolic pressure below 70 includes 25 patients with syphilis of the heart or aorta and two patients with rheumatic heart disease.

Arterial Sclerosis.—Arterial sclerosis was characteristic of these patients. In relatively few instances was an eyeground examination by a practiced ophthalmologist recorded, yet routine physical examination disclosed evidence of sclerosis in 78 per cent of the cases.

Cardiac Diagnosis.—In only 41 per cent of all cases was the principal diagnosis one relating to the heart or the aorta. Including all recorded diagnoses, both primary and secondary, a "cardiac diagnosis" occurred in only 132 cases (56 per cent).

Type of Heart Disease.—It is extremely interesting to note that of the 132 cases showing a cardiac diagnosis one hundred were cases of arteriosclerotic heart disease, hypertensive heart disease or angina pectoris; twenty-five were cases of syphilitic heart disease or aneurysm; while there were only five cases of rheumatic heart disease, one of thyroid heart disease, and one of bacterial endocarditis. In 125 of the 132 cases, then, there was more or less evidence (direct or implied) of some sort of coronary lesion.

Cases With no Cardiac Diagnosis.—Of the remaining 102 patients, those who had no cardiac diagnosis, there were only six in whose cases neither arterial sclerosis nor hypertension was diagnosed. In these six cases indeed no secondary diagnoses were recorded; the patients' ages ranged from forty-five to sixty-three years. The case histories gave evidence of sclerosis in three of the six cases; in another the blood pressure was 240/130 mm. The patients whose electrocardiograms are of the type under discussion and who give, at the same time, few clinical evidences of heart disease, have, then, almost without exception arterial sclerosis, hypertension or both.

Thus it will be seen that coronary disease or some disease which carries with it more or less suspicion of coronary involvement occurs almost without exception in the patients who presented electrocardio-

grams of this type. This is true whether examination gave enough evidence of heart disease to warrant a cardiac diagnosis or not.

Decompensation.—In 43 cases, or 18 per cent, there was a diagnosis of decompensation. Study of the histories indicated that some degree of cardiac insufficiency either had existed or was present in 88 of the remaining cases, thus bringing the total of those with past or present evidence of some degree of cardiac insufficiency to 131 cases (56 per cent).

Hypertrophy.—This was diagnosed in 110 cases, 48 per cent. In 97 additional cases it was thought likely, from the incomplete records available, that some degree of enlargement existed. An attempt to say whether or not the heart is enlarged, without complete data including the weight of the patient, the shape of the chest and a teleroentgenogram, must be, of course, only a rough estimate in many cases. It is well known, however, that cardiac enlargement is one of the most constant accompaniments of all types of chronic heart disease.

Autopsy Findings.—Unfortunately there were records of postmortem examination in only seven cases. It is notable, however, that coronary disease was found without exception in these seven instances.

Rheumatic Heart Disease.—This was conspicuous by its rarity. There were only five cases. One of these was that of a man forty-two years of age in whose case autopsy showed sclerosis of the coronary arteries.

SUMMARY OF RESULTS

Incidence.—Less than half of these patients presented themselves on account of symptoms referable to their hearts. In a considerable number of the others, however, examination and history discovered evidence of heart disease. But the total number of cases with a cardiac diagnosis (both principal and secondary) was only a little larger than the number without such a diagnosis. Only about half of the patients gave evidence of any degree of cardiac insufficiency. One factor, however, was practically constant. The incidence of these electrocardiograms was found to be confined almost exclusively to patients who had arterial disease or some other disease which brought the coronary arteries under suspicion. Investigation of their incidence, therefore, gives strong indication that coronary disease is responsible for the particular form which they assume.

RELATIONSHIP OF ETIOLOGICAL FACTORS TO THE QUESTION OF DELAYED CONDUCTION

There remains for consideration the application of the finding of coronary disease in the incidence of these records to the question of impairment of conductivity in the right limb of the A-V bundle. It

is well known, of course, that interference with the blood supply to the main stem of the A-V bundle, or to either limb, may impair conductivity in the affected part. Other lesions may produce the same result. The above findings, however, indicate that coronary disease is by far the commonest cause of such impairment in the right limb. Other considerations have an intimate bearing on the problem. The characteristic incidence of electrocardiograms showing bundle-branch block is in patients with coronary disease. It is well known that complete absence of conductivity in one branch of the bundle (bundle-branch block) occurs in the right limb with much greater frequency than in the left. Lesser defects in conductivity due to the same process would be expected to occur in somewhat the same ratio, between the right and the left limbs, as do instances of complete block. Such, indeed, appears to be the case. Investigation by one of us shows that the records in our files which might be taken to indicate impairment of conductivity in the left limb of the A-V bundle are in fact relatively quite rare among patients in the older age groups. Just how the right branch of the A-V bundle might be expected to suffer more frequently than the left branch, as a result of coronary disease, is at once apparent upon consideration of the arterial distribution to the two main limbs of the bundle. Gross[3] has shown that the right limb is invariably supplied by a branch from only one of the main coronary arteries, the left; while the septal part of the left limb of the bundle is usually (92 per cent of cases) supplied by branches from both the right and left coronaries. In only 8 per cent of cases is the septal part of the left limb supplied by a branch from only one coronary, the left. In cases of coronary disease, therefore, affecting principally the right coronary, the right limb of the A-V bundle would not suffer. Neither would the left limb suffer materially, for it always derives at least a part of its blood supply from the left coronary. In arterial disease affecting the left coronary, however, the right limb of the bundle would invariably suffer to a greater or less extent. In most such cases of left coronary disease (92 per cent) the impairment of the conducting system *would be limited to the right branch;* while in a small number of such cases (8 per cent) impairment would obtain also in the left branch of the A-V bundle. Assuming that the principal lesion of coronary disease is as likely to occur in one coronary as in the other, the ratio of incidence of conduction defects of all degrees, due to arterial disease, in the right branch of the A-V bundle to the incidence of such defects in the left branch, therefore, would be in the ratio of 92:8.

The ratio of the number of electrocardiograms indicative of right bundle-branch block to the number of records indicative of left bundle-branch block, however, is considerably higher than 92:8. This accords both with the theory of bundle-branch block as expressed by

the electrocardiogram and with the above explanation of such block. Block in one of the branches of the A-V bundle can be exhibited in the electrocardiogram only when conduction is maintained in the other branch. For if neither branch were able to transmit an impulse, the resulting electrocardiogram would exhibit, not block in one branch of the bundle, but complete A-V block. In that small group of cases (8 per cent) in which the left coronary alone supplies both the right and left branches of the bundle, instances must occur in which conduction suffers equally in the two branches. The electrocardiogram in such cases obviously will not express the defect in the left branch alone. This reduces the number of electrocardiograms which might show left bundle-branch block, and also the number which might show lesser degrees of impairment in the left branch. The result, therefore, is that the ratio of such records to those which show impairment in the right limb is lower than the arterial distribution might seem, at first thought, to indicate. In less than 8 per cent of cases of left coronary disease, then, will the electrocardiogram show defective conduction in the left bundle branch, while in more than 92 per cent of such cases will it show a conduction defect in the right branch. It follows, therefore, that not only does arterial disease offer ready explanation of impairment of conductivity, but the arterial distribution to the branches of the A-V conducting system also explains the higher incidence of this impairment in the right limb of the bundle.

Since hypertrophy (presumably of the left ventricle more than of the right) was found to be characteristic of these cases, it might be contended that these electrocardiograms express merely "left ventricular hypertrophy." We do not maintain that relative preponderance of left muscle mass is not one of the causes of left axis deviation. But it is not the only cause. And it leaves unexplained the characteristic relationship of the T-wave to the initial deflection of the ventricular complex in those instances of left axis deviation with which our paper deals. We make no attempt to offer an explanation of causes underlying the changes in electrical potential that produce the T-wave, but we emphasize the fact that in this particular type of electrocardiogram the relationship of the QRS complex to the T-wave is that of defective right bundle-branch conduction. Since this also is one of the causes of left axis deviation, and since the study of our cases indicates the occurrence of a constant factor that would be expected to impair conductivity in the right limb of the bundle, the evidence is very great that defective right bundle-branch conduction rather than some other cause of left axis deviation is responsible for the particular shape which these electrocardiograms assume.

SUMMARY AND CONCLUSIONS

The incidence of electrocardiograms showing left axis deviation and QRS of normal duration, with inverted T_1 and upright T_3, is confined almost exclusively to patients who exhibit either coronary diseases or some other disease which makes the presence of coronary involvement more or less probable.

Experimental and clinical data strongly indicate that these electrocardiograms represent impairment of conductivity in the right limb of the A-V bundle.

Consideration of the arterial distribution to the limbs of the bundle makes it appear highly probable that the conduction defect is induced by the concomitant vascular process.

Vascular lesions may be of sufficient consequence to produce such conduction defects, and electrocardiograms of this type, therefore, may be recorded before other important evidences of cardiac involvement become apparent.

The question of the influence of digitalis on the shape of these electrocardiograms, particularly so far as it concerns the inverted T-wave in Lead I, deserves comment. It is well known that digitalis produces inversion of the T-wave. It does not, however, in normal electrocardiograms, lower T_1 and, *at the same time*, elevate T_3. There is much evidence that it depresses conductivity in the bundle-branches. In cases of defective right bundle-branch conduction, therefore, such a defect might easily be increased by the drug, the characteristic electrocardiographic features being accentuated, T_1 becoming deeper and T_3 becoming higher. We have observed such results in some of our cases. Many of our patients had had digitalis at the time of their first electrocardiogram, many had not. Just how far digitalis may have accentuated the characteristic features in some of these records we have not attempted to determine. The importance of doing so would appear to bear only on the question of the extent of digitalis action on bundle-branch conduction, for it would appear extremely unlikely that the drug could *produce* such a defect in cases in which no impairment already existed. The defect in right bundle-branch conduction shown in our records, therefore, while accentuated by digitalis in some instances, perhaps, depends even in such cases primarily on a pathological process, and is not produced by the drug.

This conclusion is further strengthened by the following consideration: Digitalis had been administered, not only to the patients whose electrocardiograms we studied, but also to the patients whose records did not meet the criteria for selection, in (presumably) about the same proportion of cases. Yet our study shows that it was only in those patients with evidence of arterial disease that electrocardiograms were recorded which showed the exact relationship of QRS to T complexes indicative of right bundle-branch defect. Any primary effect of digitalis in producing the characteristic shape of these electrocardiograms, therefore, would appear to be very unlikely.

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THE PRE-EMPLOYMENT EXAMINATION OF THE CARDIAC WORKER*

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THE examination of the prospective employee made its appearance in industry some years ago and chiefly in those industries where there existed important hazards with reference to other persons outside of the business: such industries, for example, as railroads, street railway companies, operators of public conveyances, and other forms of service or industry where numbers of persons might be subject to the results of the acts of employees of the company rendering the service. From that point the examination of the prospective employee has gradually spread throughout the industrial world; doubtless receiving, in part, its impulse through the various forms of benefit and relief payment plans as well as from the various state compensation laws. And now we believe that the pre-employment examination has a definite place in industry, not as is often assumed for the purpose of eliminating the unfit, but for the purpose of a physical appraisal of the personnel so that the employer may know the physical "status quo" of the additions to his force.

Theoretically, the physical examination for employment should exclude only those members of society who through their physical condition constitute a hazard to themselves, to others or to property or service. The pre-employment examination should have for its purpose the recognition, the evaluation, and cataloguing of the several acceptable physical impairments of the individual and his allocation in the business according to the physical abilities to perform, as determined by the several impairments found. Under such a plan the number of rejections under ordinary conditions should be relatively few, and, furthermore, industry through its attitude toward the pre-employment group would be making a real contribution to public health in keeping with our present knowledge of social economics. There are times, however, when pre-employment rejections may become relatively numerous; such conditions as a very abundant labor market where it is easy to imagine that an industry in its physical examination plant would be obliged to pick and choose logically. Under such circumstances, it is only natural to expect that the standard of their physical requirements would be raised, and out of a large number of applicants selection made of those best suited physically for employment. This foregoing policy with reference to pre-employment examination ap-

*From the office of the Medical Director, American Telephone and Telegraph Company.

Read at the Annual Meeting of the American Heart Association, New York, N. Y., Feb. 4, 1929.

plies with equal force when we come to consider the cardiac applicant. It is a fact that at present in many industries, the cardiac applicant, no matter what form of disease may be present, is considered "persona non grata," idealistic pre-employment examinations notwithstanding. This possibly may be due to several factors:

First, the time element. For economic reasons, the pre-employment examination must necessarily be brief. The time allotted to the average industrial plant examination is so short that a careful study of an abnormal heart cannot be made in every instance. Thus, quite general rejections where prominent impairments of any sort are found.

Second, the ability of the examiner. It is a recognized fact that the major portion of those concerned in pre-employment physical examinations are not skilled to the degree of deciding whether or not, based on their findings, they should or should not recommend the employment of a given cardiac applicant. Thus, quite often in the industrial medical department the examiner through lack of time, plus no small degree of ignorance, fails to make his contribution.

THE COMPENSATION LAWS

Industry is not altogether to be blamed for the unfortunate attitude which it has taken toward the prospective employee with a cardiac condition. It must be admitted that employers in certain states are prejudiced against the cardiac applicant no matter what the type. As the compensation commissions go more and more into the consideration of the accident problems rising out of or in the course of employment, they see many instances where, though on doubtful bases, they feel compelled to render decisions against employers, because of what they may consider as aggravated, predisposing cardiac causes to certain accidents. If, on the other hand, the industry has a definite record of the cardiac standing of a particular employee, and at the same time has a record of the type, duration, and supervision of the employment, there is then in the possession of the employer real evidence to be presented if at any time these cases as the result of accident come to the attention of the local commissions. Such evidence should be of value to commissions and assist them in arriving at decisions as to compensation.

COMPANY OR PARTICIPATING PLANS FOR SICKNESS RELIEF

In many industries of the present day where personnel problems have been thought out along sound sociological lines, there exist plans for benefits and pensions. If individuals are accepted for employment who have cardiac impairments showing signs of inevitable catastrophe within a relatively short time, it would be poor business to expend company funds in expensive training. It would likewise hardly be justifiable to place them in line to receive sickness benefit payments. Again, it is a fact of experience that persons with certain cardiac

lesions when they become the subject of acute infectious disease, have long convalescence periods and are more liable to have complicated results.

JOB PREPARATION

In some industries before a prospective employee can qualify to a normal productive capacity, there exists a rather intensive training period. This interjects ofttimes an unlooked-for feature in employment where the effort involved may be all out of proportion to that necessary after the final job assignment is made. Meanwhile, our cardiac applicant is an employee, perhaps on probation, if you please, but nevertheless he is an unusual liability in a double sense; first, that growing out of the temporary but unusual nature of the employment and, second, because of the necessarily costly nature of the instruction during the training period.

If it were possible to accept the major portion of those suffering from cardiac disease, who are in a position to go to the employment office and apply for a job, there would be an opportunity to render a service to the community, in that the industry thereby would accept its own proportion of the community's social responsibility. It would likewise be to the advantage of the acceptable cardiac worker, because such an individual immediately would have an opportunity to gain confidence in himself, which, if he has been considered a cardiac applicant for a few months and during that time has applied for employment, he has had many opportunities to have it shaken. Furthermore, in those industries maintaining medical departments, there is presented an opportunity for medical supervision to the extent of seeing that the job allotted fits into the sphere of what is acknowledged as a proper one for the given cardiac impairment. This supervision may also be exercised to the extent of insisting that the employee at proper times consult a capable family physician or resort to a well-conducted cardiac clinic.

The cardiac employee is an economic problem and if industry maintains the same attitude of stringency toward all cardiac employees, there is going to be progressive economic loss due to wastage of utilizable talent. The statement that there are two million people with heart disease in the United States is without doubt conservative. The wartime draft examinations rejected 42.3 men out of every thousand because of heart disease. Of this large number, it would be logical to say that at least 50 per cent are employable in some capacity or other.

The cardiac individual who some time in his career has the label "cardiac" tied to him is a poor advertisement for himself. He perhaps too often becomes afflicted with a certain mental twist whereby he is naturally disinclined to do those things which he either thinks or has been told will damage his already damaged heart. As a result,

there is a timidity and a tendency toward introspection and the avoidance of physical effort which ofttimes may be out of proportion to the actual cardiac state present. He then puts himself at once in a compromising position.

In fairness to industry, cardiac employees must always be considered for special or modified employment and, when considered from the standpoint of industry, they may be roughly divided into two groups:

First, those who can work without discomfort, and second, those who cannot work without discomfort.

Under this latter heading there need be no further subdivision, as industry aside from the medical standpoint cannot and should not be concerned with them.

Under the division of those who can work, we may make a number of artificial classifications:

1. Those who can work full time.

2. Those who can work on some individually determined limited time bases.

It may be possible under those classified as full time cardiac employees to subdivide still further this group under:

a. Those capable of working under conditions of ordinary limited physical or mental strain.

b. Those capable of working only under specially determined limited physical or mental strain.

In the group of individuals working under limited time, the subdivisions may be practically the same as those classified under full time cardiac employees.

In determining the acceptability of a given cardiac applicant, either on a full time or on a limited time basis, the clinical signs necessarily center about a few easily determined factors: first, the history with reference to the cause of the cardiac condition; and second, the history with reference to the result of the cardiac condition (in other words, the compensation history).

A rather brief physical survey may show both physical and auditory signs of what seems to be a present or previous decompensation catastrophe. To make this somewhat more explicit, there is not so much determinative information to be obtained through an auscultatory examination, in view of the fact that the ordinary examiner is not in a position to form an opinion with reference to employability based on the type of lesion indicated by the cardiac sounds. Of more importance are the functional phenomena, such as shortness of breath following moderate exercise, cyanosis of the lips and ears, enlargement and pulsation of superficial vessels, peripheral edema, and other signs indicating that a decompensation is well under way or may be in the process of subsidence. In determining the employment of a

given cardiac applicant, these general physical indices should be taken into account together with the character of the reestablished compensation, in order to decide, first, whether the compensation has been brought about with no functional residues, and second, if there still exists evidence in the compensation of functional residues.

How employable a prospective employee may be, as based on compensation breaks, may in a measure, rest on the number of times and the character of each pre-existing breakdown of the compensation, together with the intervening rehabilitation time.

My own impression would be that in an individual of questionable general physique who gives a history, or in whom in the course of a physical examination there exist evidences of two or more breaks, the question of employment should be deferred, perhaps to come up for reconsideration after the lapse of a reasonable period of time. Dogmatic finality should be avoided as far as possible, as these individuals need encouragement at every turn.

The cardiac applicant should also be appraised as to the amount of psychical damage that has grown out of his experience with the cardiac impairment. Many such individuals are psychoneurotic to a pronounced degree. Such cases can become a serious drain on industry through absenteeism because of occasional real and more often imaginary indisposition. Except in instances where such individuals can have more or less solitary employment, they may become real problems to their superiors, due to the effect on the morale of the rest of the group among which they may be working. It is humanly impossible over any extended period of time, in spite of real sympathy and charity in judgment, to overcome a certain element of resentment on the part of other employees who are witnesses to these necessary breakdowns in departmental routine. Furthermore, certain cardiac workers, when the victims of some acute disease process, become a further problem because of the marked extension in convalescence time.

In planning a program of pre-employment examinations, there are a number of factors to be considered, and it must be remembered that in an industry each new departure will be conducted along definite economical lines. Throughout industry in general, the funds available for the employment of physicians as examiners will not permit medical departments to hire experts in cardiac diagnosis or, indeed, little talent that could be classed as expert. Furthermore, the question of time allotment for an examination will sooner or later loom on the horizon and as medical work grows in any given industry, the various steps of the pre-employment examination and even the periodic examination will have allotted to it a time limit, not rigid of course, but indicating to the medical employee that he likewise must produce and get his product out with a certain amount of celerity. It will thus be seen that, in the course of an average pre-employment examination,

there will probably be from five to ten minutes spent on the heart examination and then only upon evidence of unusual cardiac pathology. Thus it is easy to see that the average physical appraisal of an applicant for employment who has cardiac disease must of necessity be superficial in the extreme, because of lack of special diagnostic skill, of lack of time, and of lack, if you please, of interest in the question of the pathology present (since to the average examiner such a case is not a personal patient or even an object of much concern).

The decision for or against employment must be based on a composite cardiac picture consisting of:

1. A few hard and fast rules governing the actual heart examination.
2. A brief psychical appraisal.
3. The history or evidence of present or recent decompensation.
4. The establishment of the fact that the applicant has or has not complied with clinical requirements regarding the elimination of sites of focal infection.

Once having determined that a given applicant for employment has cardiac disease, there are certain examination facts which will decide against recommendation for employment and these may be listed as follows:

1. Inspection signs of cardiac disease and accompanying decompensation or phenomena of endocrine dyscrasia.
2. Any diastolic murmur or thrill.
3. Auricular fibrillation.
4. A rate, after rest, that is persistently above 90 or below 50 or a pulsus alternans.
5. Paroxysmal tachycardia.
6. A persistent systolic blood pressure, in adults up to forty years of age, of 160 or more, or of 100 or below after complete horizontal rest for ten minutes.

An individual with cardiac disease to be employable under a special assignment schedule, as previously stated, should present no gross inspection signs of cardiac disease or decompensation following simple exercise functional tests. The cardiac murmur present should not be transmitted into the neck. Where pulse intermittency exists, it should be the result of extrasystoles and should not be accompanied by a palpable thrill or tumultuous impulse in the area of the apex beat. The rate should be within normal limits.

These few clinical data should be easily elicited by the average man doing medical department pre-employment examinations. It is felt that the exclusion of those handicapped to the extent indicated in the nonacceptable requirements would constitute a hazard in any form of employment, from the standpoint of affecting the morale of the rest of the force, their own and others health or safety, and through the limited contribution to the product or service of the particular part of the industry in which they desired to be employed.

MORTALITY AND MORBIDITY FROM HEART DISEASE UNDER INDUSTRIAL AND GROUP INSURANCE

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HEART disease and limitation of capacity for physical exertion are so closely associated, and heart disease may be so aggravated by physical exertion that it might quite naturally be assumed that the interrelations of heart disease and occupation would be reflected in high mortality rates in at least a considerable number of occupational groups. There is little evidence, however, that such excessive occupational mortality exists.

This is a matter of particular interest when it is remembered that heart disease is foremost among the causes of death. In striking contrast stand pulmonary tuberculosis, pneumonia and accidents, all of which display clearly the influence of occupation upon mortality rates.

Tuberculosis is the cause of grossly excessive mortality among such workers exposed to silicious dusts as hard rock miners, pottery workers, stone cutters and grinders and among such workers as waiters, cigar makers, laundry workers and printers (see Tables I and VI).

Pneumonia causes a still more marked excessive mortality among iron foundry workers, cordage workers, metal polishers, miners and steel workers.

Deaths from accident are more readily associated with occupation, and the very high death rates from accidental violence among electric linemen, railway workers, coal miners, structural iron workers, city firemen, roofers and others are, of course, extreme examples of the effect of occupation upon mortality.

There is undoubtedly some degree of association of social or economic status and mortality from heart disease. This is reflected in the mortality rates from this cause in the several classes of insured persons. The "ordinary" class is made up, generally speaking, of the most favored individuals; the "industrial" class is made up largely of wage earners holding small policies, and the "intermediate" class falls between.

These observations are confirmed in the last report of the Registrar General of England and Wales, 1921-1923.

It is unfortunate that the statistical data relating to deaths among those covered by group insurance and by industrial insurance are not, at present, in such form as to permit of the recognition of mortality rates by specific occupations.

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Read at the Annual Meeting of the American Heart Association, February 4, 1929.

TABLE I

OCCUPATIONS SHOWING THE HIGHEST PERCENTAGE OF DEATHS FROM SPECIFIED CAUSES OF DEATH LISTED ACCORDING TO THEIR STANDARDIZED RATIOS*

MORTALITY EXPERIENCE OF METROPOLITAN LIFE INSURANCE COMPANY ON WHITE MALE INDUSTRIAL POLICYHOLDERS 15 TO 64 YEARS. 1922-1924

TUBERCULOSIS OF THE RESPIRATORY SYSTEM		PNEUMONIA (ALL FORMS)		ACCIDENTAL OR UNDEFINED VIOLENCE	
OCCUPATION	STANDARDIZED RATIO	OCCUPATION	STANDARDIZED RATIO	OCCUPATION	STANDARDIZED RATIO
Miners (underground) excluding coal miners	182.7	Iron foundry workers	220.0	Electric linemen	280.8
Pottery workers	176.5	Cordage and hemp mill workers	185.0	Railway enginemen and trainmen	240.0
Stone cutters	144.4	Polishers (iron and steel products)	183.8	Coal miners (underground)	227.5
Waiters and hotel servants	140.7	Coal miners (underground)	150.0	Structural iron workers	218.3
Cutlers and grinders	135.8	Iron and steel mill workers	142.5	Firemen (city department)	196.7
Cigar makers and tobacco workers	135.2	Rubber factory workers	135.0	Roofers	188.3
Laundry workers	134.6	Hucksters and peddlers	132.5	Fishermen, oystermen, sailors and marine workers	185.0
Compositors, printers and pressmen	130.9	Roofers	131.3	Miners (underground) excluding coal miners	184.2
Brass foundry workers	126.5	Longshoremen and stevedores	130.0	Railway track and yard workers	176.7
Barbers and hairdressers	125.9	Brick, tile and terra cotta workers	127.5	Chemical and explosives factory workers	173.3
Glass workers	123.5	Boiler makers	123.8	Cement and lime workers	170.0
Clerks, bookkeepers and office assistants	122.8	Glass workers	120.0	Oil refinery workers	160.8
Polishers (iron and steel products)	122.2	Laborers	120.0	Brick, tile and terra cotta workers	160.0
Shoe factory workers	118.5	Blacksmiths	120.0	Chauffeurs	145.8
Tailors and other clothing workers	118.5	Miners (underground) excluding coal miners	117.5	Watchmen and guards	145.0
Furniture and other woodworkers	118.5	Stone cutters	117.5	Saw and planing mill workers	143.3
Cordage and hemp mill workers	117.9	Street and sewer cleaners	115.0	Electricians	143.3
Slaughter and packing house workers	117.9	Masons and bricklayers	113.8	Paper and pulp mill workers	140.8
Shoemakers (cobblers)	116.7	Cement and lime workers	111.3	Longshoremen and stevedores	137.5
Bakers	116.0	Oil refinery workers	110.0	Carpenters	128.3
All occupations (excluding retired)	100.0	All occupations (excluding retired)	100.0	All occupations (excluding retired)	100.0

*The percentage of deaths from each specified cause of death in the the age period 15 to 64 years among all occupations combined was taken as 100. The standardized percentage for each cause in each occupation divided by the corresponding percentage for all occupations combined gives the standardized ratio.

Group insurance is of relatively recent development. It is written at extraordinarily low rates because there are eliminated from the transactions all possible administrative and selective procedures. Precise statements concerning the occupations of those insured are not required. In consequence the mortality which is experienced relates to industries and not to occupations. Physical examinations are not required so there is no physical selection of lives. Among those exposed to risk are individuals of varying ages, of either or both sexes, and to some extent, white or colored. It is evident that as group insurance covers employees upon the payrolls of the respective insured

TABLE II

DEATH RATES FROM OTHER DISEASES OF THE HEART
METROPOLITAN LIFE INSURANCE COMPANY, 1927
DEATHS PER 100,000 LIVES EXPOSED AT SPECIFIED AGES

AGE PERIOD	ORDINARY CLASS (MALES)	INTERMEDIATE CLASS (MALES)	INDUSTRIAL CLASS (WHITE MALES)
20 to 24 years	11.2	12.6	20.5
25 to 34 years	10.9	18.3	38.3
35 to 44 years	27.9	52.1	90.0
45 to 54 years	118.6	195.8	284.0
55 to 64 years	343.9	427.8	738.4
65 to 74 years	1013.9	1171.3	1785.8
75 years and over	3168.6	4166.7	4295.2

TABLE III

STANDARDIZED MORTALITY OF THE FIVE SOCIAL CLASSES AND OF ALL MALES. AGES 20 TO 65 YEARS
"VALVULAR DISEASE" OF THE HEART AND "OTHER HEART DISEASE"
REPORT OF THE REGISTRAR GENERAL FOR ENGLAND AND WALES, 1921-1923

	SOCIAL CLASS*					ALL MALES
	I	II	III	IV	V	
Valvular disease of heart	36.1	57.2	61.1	67.3	80.9	63.9
Other heart disease	69.7	71.6	59.0	60.4	75.6	64.9

*Social Class I—Upper and Middle.
Social Class II—Intermediate.
Social Class III—Skilled Workers.
Social Class IV—Intermediate.
Social Class V—Unskilled Workers.

groups, there is a tendency to eliminate from the exposure those individuals whose incapacity because of heart disease is such as to prevent them from engaging in active work. A portion of those incapacitated because of heart disease may, under group insurance, become eligible for total and permanent disability benefits. Such cases are charged as claim losses as of the dates of acceptance of the claims. Unfortunately, published statements of group claim experience do not offer a classification of such permanent and total disability claims, by cause. About 15 per cent are due to heart disease.

Accepting such data as are available in connection with the mortality from heart disease among the several million workers covered under group insurance, it is apparent that there exist relatively unim-

portant differences between the major classifications, such as "mining," "iron, steel and other metals," "transportation," "clerical," and "all others." Such differences as are observable in the several rates for each age are for the greater part to be explained by elements of selection incident to the demands of the respective industrial classifications. The cardiac cripple will, for example, be almost automatically excluded from a heavy occupation. The operation of such tendencies has evidently been responsible for the levelling of cardiac mortality rates among the various occupational classes.

TABLE IV

DISEASES OF HEART AND CIRCULATORY SYSTEM
DEATHS PER 100,000 LIFE YEARS

CENTRAL AGE	MINING	IRON, STEEL OTHER METALS	TRANSPORTATION	CLERICAL	ALL OTHERS
	Class I	Class II	Class III	Class IV	Class V
18	14.0	6.2	16.6	19.8	21.3
23	23.8	26.8	23.8	13.8	21.5
28	34.6	31.6	29.7	28.4	25.6
33	46.8	45.9	39.7	32.8	38.5
38	45.2	73.9	76.6	53.2	65.5
43	112.9	122.8	133.2	95.9	111.5
48	188.3	177.1	229.1	184.7	185.1
53	316.5	344.9	361.3	356.2	296.8
58	505.7	529.6	594.2	546.6	515.7
63	765.6	816.6	1020.2	1000.0	872.9
68	1325.3	1600.0	1774.3	1551.1	1386.2
73	1293.1	2552.6	2556.6	2394.7	1956.6
78	2991.4	4354.8	4655.1	4114.3	3273.7
83	3333.3	6611.5	6666.6	5431.3	6257.2
88	5263.1	10000.0	9230.7	3571.4	7260.6

At the present time group insurance data concerning morbidity from heart disease in relation to occupation are not to be had. It is possible, however, to present some general figures indicating the relative importance of diseases of the circulatory system among the many causes of illness for which benefits are payable in time of sickness.

In a study of 6671 claims incurred under policies paying benefits for approximately twenty-six weeks beginning on the eighth day of illness among white males, a life year exposure of 173,000 years, there were 323 or 4.8 per cent of the total number paid for losses due to various types of circulatory diseases, about 2 per 1,000. These claims involved the payment of 9 per cent of the total benefits paid for all causes and were of an average duration of 75.7 days which may be compared with an average duration of benefit of 41 days, and which is a greater duration than that associated with any other group of disabilities.

Among white females under similar policy provisions, 34,000 life years, the diseases of the circulatory system accounted for 32 claims out of 1154 or 2.8 per cent of the total, about 1 per 1,000; on these claims were paid 5.9 per cent of all benefits paid; and the average duration of benefit was 80.8 days which may be compared with an

average duration of benefit of 44 days. The average duration of disability due to circulatory diseases in this instance also was greater than that due to any other cause.

In view of the many circumstances which would incline the vital statistician to view group insurance mortality data as treacherous and untrustworthy, it is of interest that an extensive mortality experience among men covered by that form of insurance known as industrial insurance shows an almost identical picture.

These data relate to deaths among individual policyholders whose occupations at death are recorded. Unfortunately, the number of lives at risk in each occupational classification cannot be obtained, so true death rates are not available. In consequence it is necessary to resort to the use of a form of statement of mortality, which, while useful and truly informative, leaves much to be desired. The proportion of deaths from any cause, such as heart disease, in an occupational group is corrected for age and expressed in ratio to the proportion of deaths from that cause among all occupied males. This figure is known as the "standardized ratio."

In a study recently undertaken by the Statistical Bureau of the Metropolitan Life Insurance Company, under the direction of Dr. Louis I. Dublin, there was determined the relation of occupation to 105,467 deaths which occurred during the three years, 1922 to 1924, among three and one quarter million male white industrial policyholders.

The great majority, 90 per cent, of cardiac deaths were due to the cause of the international classification known as "Other Diseases of the Heart," heart disease excluding pericarditis, acute endocarditis, acute myocarditis, and angina pectoris.

TABLE V

NUMBER OF DEATHS IN SPECIFIED AGE PERIODS FROM ALL CAUSES AND FROM "OTHER DISEASES OF THE HEART." MORTALITY EXPERIENCE OF METROPOLITAN LIFE INSURANCE COMPANY ON WHITE MALE INDUSTRIAL POLICYHOLDERS (EXCLUDING RETIRED) 15 YEARS AND OLDER, 1922 TO 1924

CAUSES OF DEATH	NUMBER OF DEATHS						
	AGES 15 YEARS AND OVER	15 TO 24 YEARS	25 TO 34 YEARS	35 TO 44 YEARS	45 TO 54 YEARS	55 TO 64 YEARS	65 YEARS AND OVER
All causes	105,467	11,927	12,153	15,041	19,656	25,054	21,636
Other diseases of the heart	16,217	903	854	1,386	2,982	4,941	5,151

To this cause, "Other Diseases of the Heart," were assigned 16,217 deaths of individuals fifteen years or over, and it is to this class that the following data relate. The standardized ratios were computed for those deaths between the ages of fifteen and sixty-four years.

With the "ratio" of 100 for all occupied males, those for specific occupations ranged, strangely enough, from 180 among sailors of the navy to 37 among soldiers. These ratios may be ignored, however, as they

TABLE VI

PERCENTAGE OF DEATHS DUE TO "OTHER DISEASES OF THE HEART" OF DEATHS FROM ALL CAUSES IN SPECIFIED OCCUPATIONS MORTALITY EXPERIENCE OF METROPOLITAN LIFE INSURANCE COMPANY ON WHITE MALE INDUSTRIAL POLICYHOLDERS 15 YEARS AND OLDER. 1922 TO 1924.

OCCUPATION OR INDUSTRY	DEATHS (15 YEARS AND OLDER)		PERCENTAGE OF DEATHS FROM OTHER DISEASES OF THE HEART		STANDARDIZED RATIO*
	ALL CAUSES	OTHER DISEASES OF THE HEART	CRUDE FOR AGES 15 YEARS AND OLDER	STANDARDIZED FOR AGES 15 TO 64 YEARS	AGES 15 TO 64 YEARS
All occupations combined (excluding retired)	105,467	16,217	15.4	13.2	100.0
Sailors (navy)	218	10	4.6	23.8	180.3
Boiler makers	367	64	17.4	17.0	128.8
Merchants and storekeepers	3,133	578	18.4	16.9	128.0
Roofers	179	26	14.5	16.3	123.5
Cooks (hotel and restaurant)	324	59	18.2	16.3	123.5
Tailors and other clothing workers	1,175	232	19.7	16.2	122.7
Textile dyeing, bleaching, and finishing mill workers	391	81	20.7	15.5	117.4
Waiters and hotel servants	789	127	16.1	15.4	116.7
Clerks, bookkeepers and office assistants	5,140	749	14.6	15.2	115.2
Barbers and hairdressers	768	132	17.2	14.8	112.1
Shoemakers (cobblers)	666	145	21.8	14.7	111.4
Hostlers and stablemen	363	77	21.2	14.7	111.4
Brick, tile and terra cotta workers	199	36	18.1	14.6	110.6
Hucksters and peddlers	932	171	18.3	14.6	110.6
Plasterers	354	62	17.5	14.5	109.8
Janitors and building employees	2,243	446	19.9	14.5	109.8
Store clerks and salesmen	3,302	490	14.8	14.4	109.1
Agents, canvassers and salesmen	1,296	212	16.4	14.1	106.8
Tinners (shop) and tinware workers	467	84	18.0	14.1	106.8
Longshoremen and stevedores	594	96	16.2	14.0	106.1

*The percentage of deaths (13.2) shown in column 4 for all occupations combined was taken as 100. The standardized percentage for each occupation divided by 13.2 gives the *Standardized Ratio.*

are based upon small numbers of deaths and being corrected for age do not accurately reflect the actual incidence of cardiac deaths in these two highly selected occupational groups.

These ratios, it must be clearly understood, are not death rates. They reflect only the importance of cardiac mortality in relation to other causes. Such indices, however, are of value in disclosing any tendency of occupation to induce an excessive mortality from any cause or causes.

The best extensive occupational mortality data of recent date now available are to be found in the report of the Registrar General of England and Wales for the period 1921-1923.

Among 164 occupational classes of those males dying between the ages twenty and sixty-five years but 16 classes showed significant standardized mortality rates from valvular diseases of the heart in excess of that for all occupied and retired males, and 21 classes, an excessive mortality from "Other Diseases of the Heart."

In only a few classes was the excess mortality at all marked. It was for other diseases of the heart particularly notable in those classes exposed to a silica hazard and with bartenders. The association of heart disease and a silica hazard was not evident in the American data.

It is possible, even probable, that there is in this country a higher degree of mobility of labor than in Great Britain, a greater tendency of workers to shift from one form of employment to another more congenial or better suited to physical capacity.

It may thus be seen that whatever may be the importance of the part played by occupation in the development or aggravation of cardiac incapacity in particular, individual cases, there is no considerable evidence that certain occupations are conducive to high death rates in the battle with the new captain of the men of death.

STATISTICAL STUDIES BEARING ON PROBLEMS IN THE CLASSIFICATION OF HEART DISEASES

V. Heart Disease Among Ex-Service Men*†

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(Continued from page 313, February issue)

DEATHS FROM CARDIOVASCULAR DISEASE

THE data received from the hospitals indicated that 60 beneficiaries died from cardiovascular disease. It was thought that it might prove of value to analyze these deaths for the purpose of ascertaining the etiological factor of the heart disease, the age of the patient at death, and the specific lesion or lesions causing death. These facts are brought out in Tables XIV, XV, and XVI, which follow:

Table XIV lists a series of 60 patients who had been under treatment for various cardiovascular diseases and who died. The cardiovascular lesions are arranged according to etiology as well as to specific diagnosis. These 60 patients gave a history of having had 100 cardiovascular lesions, the latter being due to 116 factors. It is thus seen that the average number of lesions per patient was 1.6 and the average number of factors per patient was 1.9.

Thirty-four, or 29.31 per cent, of the lesions were of rheumatic origin; 21, or 18.10 per cent, were sequelae of infectious diseases; 14, or 12.07 per cent, were syphilitic; and in 37, or 31.90 per cent, of the lesions, the etiological factor was unknown.

Of the total number of lesions, namely 100, the most frequent were: chronic myocarditis, enlargement of the heart, aortic insufficiency, mitral insufficiency, and mitral stenosis, in the order named.

The 60 patients referred to in Table XIV are again listed in Table XV so as to indicate the direct cause of death as well as the age at death. It is noted that 32, or 53 per cent, of the patients died from organic disease of the heart; 13 from pulmonary tuberculosis; and 4 from acute endocarditis.

While, in a number of cases, the cardiovascular disease was not the direct cause of death, in 32, or 53 per cent, of the group the disease of the heart was the primary cause of death. It is probable that in those

*From the Research Subdivision Medical Service, U. S. Veterans Bureau, and from the Research Committee of the Heart Committee of the New York Tuberculosis and Health Association.

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TABLE XIV

DEATHS OF 60 CARDIAC PATIENTS, BY SPECIFIC DISEASE, AND BY ETIOLOGICAL TYPE OF HEART DISEASE

SPECIFIC DISEASE	RHEUMATIC	SEQUELAE OF INFECTIOUS DISEASES	SYPHILITIC	TOXIC	GENERAL SYSTEMIC DISEASE	THYROID	OTHERS	UNKNOWN	TOTAL ETIOLOGICAL FACTORS	TOTAL HEART LESIONS
Myocarditis, chronic	6	3	1	3	1			4	18	14
Enlargement of heart	3	4						8	15	14
Cardiac valvular disease, aortic insufficiency	6	2	3					3	14	13
Cardiac valvular disease, mitral insufficiency	1	2	2					7	12	12
Cardiac valvular disease, mitral stenosis	6	2				1		2	11	10
Hypertrophy of heart	1	2			1			4	8	7
Aortitis, without dilatation	1		4						5	5
Endocarditis, acute	2						1	1	4	4
Thrombosis of femoral, saphenous and popliteal veins		2					1		3	1
Auricular fibrillation		2						1	3	3
Heart-block	2	1							3	1
Arrhythmia	2	1							3	1
Myocarditis, acute	1			1					2	1
Fatty degeneration of heart								2	2	2
Arteriosclerosis, general			1					1	2	2
Cardiac valvular disease, aortic stenosis	1		1						2	2
Hypertension	1				1				2	1
Fatty infiltration of heart								1	1	1
Tumor of pericardium			1						1	1
Aneurysm of aorta			1						1	1
Aneurysm of pulmonary artery								1	1	1
Aneurysm of ulnar artery	1								1	1
Thrombosis of veins of pelvis and legs								1	1	1
Endocarditis, chronic								1	1	1
Total	34	21	14	4	3	1	2	37	116	100
Per cent	29.31	18.10	12.07	3.45	2.59	0.86	1.72	31.90	100.00	

TABLE XV

DEATHS OF 60 CARDIAC PATIENTS BY DIRECT CAUSE AND AGE AT DEATH

DIRECT CAUSE OF DEATH	22-23	24-25	26-27	28-29	30-31	32-33	34-35	44-45	46-47	50-	AGE NOT STATED	TOTAL
Organic diseases of the heart		2	2	1	4	1		1	2	2	17	32
Tuberculosis of the lungs	1	1	1	1	3	1	3				2	13
Endocarditis, acute		2									2	4
Hemorrhage; other diseases of the circulatory system							1			1	1	3
Diseases of the arteries, atheroma, aneurysm, etc.											2	2
Bright's disease											2	2
Pott's disease				1								1
Cancer and other malignant tumors of other organs or of organs not specified						1						1
Pulmonary congestion, pulmonary apoplexy											1	1
Diseases of the stomach (cancer excepted)											1	1
Total	1	5	3	3	7	3	4	1	2	3	28	60

TABLE XVI

SHOWING THE PRIMARY CARDIOVASCULAR DISEASE FOR WHICH 60 EX-SERVICE PATIENTS WERE HOSPITALIZED AND WHO DIED DURING HOSPITAL STAY

CARDIOVASCULAR DISEASES	AGE OF DEATH											
	22-23	24-25	26-27	28-29	30-31	32-33	34-35	44-45	46-47	50-	NOT STATED	TOTAL
Myocarditis, chronic, fibrous					2			1	1	1	1	6
Enlargement of heart			1		1	1	1					4
Endocarditis, acute		1					1				2	4
Mitral stenosis					1					1	2	4
Aortic insufficiency						1					2	3
Mitral insufficiency and mitral stenosis									1		2	3
Enlargement of heart and hypertrophy of heart					1		1					2
Enlargement of heart and myocarditis, chronic, fibrous				1							1	2
Enlargement of heart and fatty degeneration	1					1						2
Myocarditis, chronic, fibrous and auricular fibrillation			1				1					2
Aortic insufficiency and aortic stenosis				1							1	2
Mitral insufficiency											2	2
Enlargement of heart, hypertrophy of heart, and myocarditis, chronic, fibrous											1	1
Enlargement of heart and chronic endocarditis											1	1
Enlargement of heart and aortic and mitral insufficiency			1									1
Enlargement of heart and mitral insufficiency					1							1
Hypertrophy of heart, myocarditis, chronic, fibrous, and fatty infiltration											1	1

TABLE XVI—CONT'D

CARDIOVASCULAR DISEASES	AGE OF DEATH											
	22-23	24-25	26-27	28-29	30-31	32-33	34-35	44-45	46-47	50-	NOT STATED	TOTAL
Hypertrophy of heart, myocarditis, chronic, fibrous, and hypertension											1	1
Hypertrophy of heart and aortic insufficiency											1	1
Hypertrophy of heart and mitral insufficiency		1										1
Myocarditis, acute											1	1
Myocarditis, chronic, fibrous, heart-block and arrhythmia										1		1
Tumor of pericardium and aortitis without dilatation											1	1
Aortitis and aneurysm of aorta											1	1
Aortitis and arteriosclerosis, general											1	1
Aortitis and aortic insufficiency		1										1
Aortitis and aortic and mitral insufficiency											1	1
Aneurysm of pulmonary artery		1										1
Aneurysm of ulnar artery, aortic insufficiency, and mitral stenosis		1										1
Thrombosis of femoral, saphenous, and popliteal veins											1	1
Thrombosis of veins of pelvis and legs				1								1
Arteriosclerosis, general											1	1
Aortic and mitral insufficiency											1	1
Aortic insufficiency and mitral stenosis											1	1
Mitral insufficiency, heart-block and arrhythmia					1							1
Mitral stenosis and auricular fibrillation											1	1
Total	1	5	3	3	7	3	4	1	2	3	28	60

cases where death was not directly due to cardiovascular disease, the latter might have been a contributing factor.

Table XVI is a compilation of the 60 cases referred to in Tables XIV and XV arranged according to the specific cardiovascular disease for which they were hospitalized and according to the age at death. Attention is again invited to the fact that even though cardiovascular disease existed at the time, it was not necessarily a primary cause of death, but may have been a contributory factor.

It has been pointed out by Dublin, and also by Cohn, that five-sixths of the deaths from heart disease take place after the age of forty, while only one-sixth of the deaths occur prior to that age.

The data in Table XVI indicate that of the total of 60 cases the age at death of 28 is not stated. In the series of 32 cases, in which the age at death was recorded, it is noted that 6 were between forty-four and fifty years of age; 26 between the ages of twenty-two and thirty-five; 7 died at the age of thirty or thirty-one; 4 died at the age of thirty-four or thirty-five; and 5 died at the age of twenty-four or twenty-five. These figures are not of particular significance in view of the fact that the group as a whole is a select one and the average age of these patients is approximately thirty-six years.

A study of the specific heart lesions for which these patients were hospitalized indicates that 25, or 41.67 per cent, of the patients had some chronic valvular disease of the heart.

Mortality Rate From Cardiovascular Disease in the U. S. Veterans Bureau, Fiscal Years 1922-1926, Inclusive.—An attempt was made to analyze the deaths in the Veterans Bureau from cardiovascular disease during the period, 1922 to 1926. Reference to Table XVII shows that the number has increased from year to year with the exception of 1924, in which year there was a material decrease in deaths due to this cause. There were 98 deaths in 1922, 192 in 1923, 147 in 1924, 206 in 1925, and 296 in 1926. The total deaths in Bureau and other hospitals from cardiovascular disease for these years was 939. The total deaths from all causes for the same period was 12,718, making the percentage of deaths due to cardiovascular disease for this period 7.38.

These figures may be compared with the observations of J. S. Whitney[10] who found that the mortality from heart disease in the civil population was 15 per cent of all deaths every year.

An attempt was made to ascertain the primary cause of death among the 939 patients dying from cardiovascular disease during 1922-1926, inclusive. Reference to Table XVIII indicates that in 661, or 70.4 per cent, of the cases the primary cause of death was organic disease of the heart.

Age at Death.—J. S. Whitney[10] found that the mortality rate of cardiac patients of the civil population is least at the ages from twenty to twenty-four years, and highest at the ages from seventy-five to seventy-nine. Dublin[2] states that heart disease is preëminently a condition of the older ages of life; but it is by no means to be neglected as a cause of death in the early years. In 1924 the Metropolitan Life Insurance Company recorded close to 20,000 deaths from heart disease among its policy holders. Of this number, 1600, or 8 per cent, were persons under the age of twenty-five years, and 3400, or 17 per cent, were under the age of forty.

TABLE XVII

YEAR	TOTAL DEATHS	DEATHS FROM CARDIO-VASCULAR DISEASE	PER CENT
1922	1299	98	7.54
1923	2639	192	7.28
1924	2502	147	5.88
1925	2866	206	7.19
1926	3412	296	8.68
Total	12718	939	7.38

TABLE XVIII

DEATHS FROM CARDIOVASCULAR DISEASES, 1922-1926, INCLUSIVE

DISEASE	1922	1923	1924	1925	1926	TOTAL
Organic disease of the heart	60	135	98	158	210	661
Acute endocarditis	7	18	6	14	19	64
Embolism and thrombosis	7	16	16	7	18	64
Hemorrhage and other diseases of the circulatory system	21	13	11	6	10	61
Diseases of the arteries, atheroma, aneurysm, etc.	1	2	6	16	26	51
Pericarditis	2	4	6	2	10	24
Angina pectoris		4	1	1	1	7
Diseases of the lymphatic system (lymphangitis, etc.)			2	2	1	5
Diseases of the veins (varices, hemorrhoids, phlebitis, etc.)			1		1	2
Total	98	192	147	206	296	939

Cohn[1] refers to a group of 954 deaths due to heart disease of which number 93.2 per cent were forty years old and over. This observer maintains that the increased rate of death from heart disease involves persons over the age of forty. Under this age the mortality rate has fallen. Cohn avers that the increased death rate from cardiovascular disease in persons over forty is predominantly due to senescent changes.

A study of Table XIX reveals the fact that of the total of 939 deaths due to cardiovascular disease, occurring during 1922-1926 among U. S. Veterans Bureau beneficiaries hospitalized in government and civil hospitals, 313 took place before the age of thirty; 438 oc-

curred within the age group from thirty to thirty-nine; 99 deaths occurred within the age group from forty to forty-nine; 43 deaths occurred within the age group from fifty to fifty-nine; and 46 deaths occurred after the age of sixty. It is thus seen that 46.6 per cent of the total number of deaths occurred within the age group from thirty to thirty-nine. The percentage of deaths from then on became less in the various succeeding age groups. Further study of the statistics in Table XIX reveals the fact that 751 of the total number of 939 patients died before the age of forty, while 188 died after that age.

This is not in accordance with the customary experience in civil life where the death rate from heart disease increases with age, but is to be expected in view of the fact that the average age of the ex-service man is thirty-six, and the age at death of most of these men falls within the various age groups twenty to thirty-nine.

TABLE XIX

DEATHS OF U. S. VETERANS BUREAU PATIENTS IN HOSPITALS FROM CARDIOVASCULAR DISEASE BY AGE AND PRIMARY CAUSE OF DEATH, FISCAL YEARS 1922-1926 INCLUSIVE

AGE AT DEATH	CARDIOVASCULAR DISEASE AS A PRIMARY CAUSE OF DEATH									
	ORGANIC DISEASES OF THE HEART	ACUTE ENDOCARDITIS	EMBOLISM AND THROMBOSIS	HEMORRHAGE, OTHER DISEASES OF THE CIRCULATORY SYSTEM	DISEASES OF THE ARTERIES, ATHEROMA, ANEURYSM, ETC.	PERICARDITIS	ANGINA PECTORIS	DISEASES OF THE LYMPHATIC SYSTEM (LYMPHANGITIS, ETC.)	DISEASES OF THE VEINS (VARICES, HEMORRHOIDS, PHLEBITIS)	TOTAL
Under 20	1			1						2
20-21	7	1	2							10
22-23	13	4		4		2				23
24-25	32	10	2	5	2	2	1			54
26-27	62	5	8	10	1	3				89
28-29	100	10	8	10	3	2	1	1		135
30-31	78	10	12	5	7	3	1			116
32-33	80	6	6	8	4	2		2	2	110
34-35	67	6	9	4	1	2	1	1		91
36-37	49	5	9	3	2	1				69
38-39	36	2	3	2	6	3				52
40-41	14	1		1	1	1	1	1		20
42-43	8		1	2		1				12
44-45	12	1	1		2	1	2			19
46-47	22			1	1					24
48-49	17		2	1	3	1				24
50-51	8				1					9
52-53	10			1	1					12
54-55	2	1		1	3					7
56-57	6		1		1					8
58-59	5	1			1					7
60-61	9			2	1					12
62-63	3				2					5
64-65	1				2					3
Over 65	19	1			6					26
Total	661	64	64	61	51	24	7	5	2	939

Deaths From Organic Disease of the Heart.—In view of the fact that 661 of the 939 deaths were due to organic heart disease it was felt that an analysis should be made of these deaths, classifying them according to the specific diagnosis and according to the age at death.

It is found that of the 661 cases the cause of death in 128 was chronic myocarditis; in 120, mitral insufficiency; in 99, combined aortic and mitral lesions; and in 82, acute cardiac dilatation.

Further study of Table XX indicates that 524, or 79.2 per cent, of the 661 deaths occurred before the age of forty, and 137, or 20.8 per cent, occurred after that age. The explanation for this has already been given in the discussion of Table XIX.

In reviewing the data compiled in Table XX it is noted that chronic myocarditis constitutes the principal cause of death from organic disease of the heart, followed by mitral insufficiency, combined aortic and mitral disease, acute cardiac dilatation, valvular heart disease, unclassified, aortic insufficiency, and mitral stenosis, in the order named.

Deaths From Cardiovascular Disease of Insured U. S. Veterans Bureau Beneficiaries.—A study was made of the mortality statistics of insured U. S. Veterans Bureau beneficiaries for the purpose of ascertaining the cause of death and the average age at death of ex-service men who had had cardiovascular disease. It was found that of 1845 deaths due to cardiovascular disease, mitral insufficiency was the cause 498 times; acute cardiac dilatation, 324 times; cerebral hemorrhage, 302 times; chronic myocarditis, 216 times; acute endocarditis, 179 times; combined aortic and mitral disease, 122 times; acute myocarditis, 104 times, and chronic endocarditis, 100 times.

Two hundred and fifty-two of the total number of 1845 insured beneficiaries were studied for the purpose of ascertaining the average age at death. It was found that the average age at death of those with acute cardiac dilatation was thirty-four years; acute endocarditis, thirty-two years; chronic endocarditis, thirty years; cerebral hemorrhage, thirty-seven years; acute myocarditis, thirty-two years; chronic myocarditis, thirty-three years; combined aortic and mitral disease, thirty-four years, and mitral insufficiency, thirty-five years.

ECONOMIC ASPECT OF HEART DISEASE

Awards for compensation are based upon the percentage evaluation of the average impairments of earning capacity resulting from the disability "in civil occupations similar to the occupation of the injured man at the time of enlistment." Impairment in ability to secure employment is also considered.

TABLE XX

CLASSIFICATION OF DEATHS FROM HEART DISEASE BY SPECIFIC DIAGNOSIS AND AGE

AGE AT DEATH	ORGANIC DISEASE OF THE HEART														
	MYOCARDITIS, CHRONIC	VALVULAR HEART DISEASE, MITRAL INSUFFICIENCY	VALVULAR HEART DISEASE, COMBINED LESIONS, AORTIC AND MITRAL	DILATATION, CARDIAC, ACUTE	VALVULAR HEART DISEASE, OTHERWISE UNCLASSIFIED	VALVULAR HEART DISEASE, AORTIC INSUFFICIENCY	VALVULAR HEART DISEASE, MITRAL STENOSIS	ENDOCARDITIS, CHRONIC	VALVULAR HEART DISEASE, AORTIC STENOSIS	DILATATION, CARDIAC, CHRONIC	CARDIAC HYPERTROPHY AND DILATATION	ANEURYSM, AORTIC	CARDIAC HYPERTROPHY	VALVULAR HEART DISEASE, TRICUSPID LESIONS	TOTAL
Under 20						1									1
20-21	1	1		1	3			1							7
22-23	1	2		2	2	3	1	1		1					13
24-25	2	7	5	3	5	3	1	4	1		1				32
26-27	5	9	14	13	12	5	2	1	1		2				62
28-29	9	21	12	17	12	8	12	7				1			100
30-31	16	24	9	4	9	7	5		1	2					78
32-33	13	11	13	10	10	7	5	8	1	1			1		80
34-35	15	10	12	11	7	6		3	2		1				67
36-37	9	6	9	7	5	6	1	2	1			2		1	49
38-39	11	8	6	4	4	1	2								36
40-41	4	3	2	1	1	2	1								14
42-43	1	1	1	1	2	1		1							8
44-45	2	3	4			2			1						12
46-47	8	3	4		2	3			1		1				22
48-49	5	1	2	1	2	1	2	1		1		1			17
50-51	3	1		1	1	1	1								8
52-53	4		1	1		2		1		1					10
54-55		1		1											2
56-57	4			1		1									6
58-59	3			2											5
60-61	4	1	1			2			1						9
62-63	1	2													3
64-65		1													1
Over 65	7	4	4	1	1	1		1							19
Total	128	120	99	82	78	63	33	31	10	6	5	4	1	1	661

Most diagnoses of heart disease were made by designated examiners employed by the Bureau, though a considerable number of such diagnoses were made by heart specialists on duty in the regional office dispensaries of the Bureau.

Observation of the types of heart examinations made by designated examiners in the field indicated such diversity of methods, particularly in exercise tests, that the Bureau prescribed a standardized heart examination for uniform employment by field examiners.[11]

The Rating of Disabilities From Diseases and Injuries.—In accordance with the provisions of Section 202, Subsection 4, World War Veterans Act, approved June 7, 1924, a schedule, taking cognizance of the occupation of the injured veteran at the time of enlistment, was prepared by the United States Veterans Bureau.

Prior to the said provision of law, the Schedule of Disability Ratings of the United States Veterans Bureau was based solely upon averages, with no recognition of occupation of the individual claimant. The preparations of the Schedule of Disability Ratings, under the terms of the Act of June 7, 1924, required pioneering effort on the part of the United States Veterans Bureau. There were few, if any, precedents available upon which to base the necessarily highly complicated and technical evaluations of disabilities as influenced by the extraordinarily large number of occupations in the United States.

The Schedule of Disability Ratings now in use by the United States Veterans Bureau and put into effect January 1, 1926, consists of two tables, which are independent and interdependent: Table I, a list of Occupational Ratings, and Table II, of Disability Ratings.

Table I is an alphabetically arranged list of occupations, a representative cross-section of the various activities in agriculture, mining, commerce, professions, etc., of American life. There are one thousand of these selected occupations. Opposite each occupation appear seventeen variants, representing various body parts, indicating estimates of the relative functional importance of the body part concerned, with respect to the occupation in question.

The diseases of the heart and pericardium are included under the caption "Chest," general arteriosclerosis comes under the caption "Systemic"; while localized arteriosclerosis would fall under the part of the body affected, as for instance chest, leg, arm, etc.

If the established occupation of the claimant at the time of enlistment does not appear in the list, an inquiry is made of the claimant regarding the type and functional requirements of his occupation, and the examiner selects a similar occupation from the list.

Table II consists of a list of injuries and diseases arranged, to facilitate reference, into separate tables, as follows:

1. Amputations, fractures, deformities, and their sequelae
2. Dental and oral
3. Eye, ear, nose, and throat
4. General medicine
5. Neuropsychiatric
6. Respiratory system and tuberculosis, pulmonary and other forms
7. Surgical
8. Combined table

Opposite the list of diseases and injuries there appear a series of nine occupational variants which are determined by and are carried over from the ratings in Table I.

Use of the Tables.—Since the introduction of this Schedule of Ratings in 1926, it has worked satisfactorily in the adjudication of compensation claims of disabled ex-service men.

The regular members of the Regional Rating Board, in cooperation with the Adjudication Division of the regional office, determine the occupation of the claimant at the time of enlistment, before the medical rating is assigned. The occupation given in the record of the Adjutant General's Office or the Bureau of Medicine and Surgery of the United States Navy is accepted.

The occupation at the time of enlistment having been satisfactorily determined, the disease or injury is next ascertained. Thereupon the medical examiner first refers to Table I, locating the appropriate occupation. The disease or injury in the case is then considered with regard to the injury variants. The figures occurring at the reading junction of the occupations with the applicable injury variants are references to the occupational variants (1-9) in Table II. Turning to Table II, the medical examiner reads and assigns the percentage of disability occurring at the reading junction of the injury list and the column reference (from 1-9) given in Table II.

Multiple disabilities are combined in accordance with the instructions in the Combined Rating Table of the Schedule.

In the adjudication of the claims for compensation the following terms are used to signify the extent of disability as well as the degree of impairment:

1. Temporary Partial (T. P.)—a disability of a temporary nature in which the degree of impairment of earning capacity may be from 10 per cent up to and inclusive of 99 per cent.

2. Permanent Partial (P. P.)—a disability of a permanent nature in which the degree of impairment of earning capacity may be from 10 per cent up to and inclusive of 99 per cent.

3. Temporary Total (T. T.)—a disability of a temporary nature in which condition there is 100 per cent of disablement.

4. Permanent Total (P. T.)—a disability of a permanent nature in which the disablement is equivalent to 100 per cent.

5. Double Permanent Total (D. P. T.)—any combination of those disabilities which entitle the beneficiary to an award for permanent total disability.

COMPENSATION FOR CARDIOVASCULAR DISEASE

Table XXI shows the number of veterans who are at the present time receiving compensation for cardiovascular disease incurred while in the military service together with the specific disease for which compensated.

In reviewing Table XXI it is noted that 16,189 beneficiaries are receiving compensation for cardiovascular disease as of March 31, 1927. The total compensable load consists of 239,433 beneficiaries. The per cent of the cardiovascular cases to the total is therefore 6.76.

The average monthly compensation of the cardiovascular cases varies from a low of $10.00 to a high of $110.00; the average for the whole group of 16,189 cases is $35.59 a month, as compared with the average monthly compensation for all beneficiaries, which is $44.55.

The annual outlay of the government for compensation for cardiovascular disease is $6,914,880.00; the total annual cost to the government for compensation for all disabilities is $127,993,800.00. The outlay for compensation for cardiovascular diseases is therefore 5.4 per cent of the total amount expended annually for the compensation of all classes of disabilities.

It is also noted in Table XXI that 5,822, or 35.96 per cent, of a total of 16,189 beneficiaries receiving compensation for cardiovascular disease are reported to have mitral insufficiency. It is believed that the incidence of mitral insufficiency in the Veterans Bureau is too high, and it is thought that the cause for it is the use of erroneous criteria in the diagnosis of this heart lesion. The mere presence of a systolic murmur at the apex does not mean mitral insufficiency. It is therefore possible that a number of the beneficiaries with this diagnosis are really cases of so-called functional heart disease. While it is probable that these patients have an occupational handicap, it is thought that, as a result of a favorable prognosis, physical and economic rehabilitation are possible.

Further study of Table XXI shows the principal cardiovascular diseases for which the disabled ex-service men are receiving compensation. The largest groups are, in the order named: mitral insufficiency, chronic myocarditis, neurocirculatory asthenia, aortic and mitral disease, mitral stenosis, aortic insufficiency, tachycardia, cardiac hypertrophy, chronic phlebitis, chronic endocarditis, hypertension, and aortic stenosis.

Table XXII is so arranged as to show the extent of disability for cardiovascular disease, the degree of handicap on a percentage basis, the number of beneficiaries within each per cent group, the average monthly payment, and the annual outlay for compensation for each group.

It is noted that 8,661, or 53.50 per cent, of the total of 16,189 beneficiaries are being compensated on a temporary partial basis. The compensation status of these men is of a temporary nature and may be changed after any physical examination conducted by the Veterans Bureau medical officers.

TABLE XXI

SHOWING THE NUMBER OF VETERANS RECEIVING COMPENSATION FOR CARDIOVASCULAR DISEASE, TOGETHER WITH SPECIFIC DIAGNOSIS, AVERAGE MONTHLY COMPENSATION AND TOTAL ANNUAL OUTLAY FOR COMPENSATION FOR EACH SPECIFIC DISEASE

CARDIOVASCULAR DISEASE	NUMBER OF BENEFICIARIES	AVERAGE MONTHLY COMPENSATION (DOLLARS)	TOTAL ANNUAL OUTLAY FOR COMPENSATION (DOLLARS)
Adherent pericardium	1	60.00	720.00
Aortic aneurysm	50	91.80	55,080.00
Angina pectoris	42	80.12	40,380.00
Aortitis	87	33.91	35,400.00
Arteriosclerosis, local	2	87.50	2,100.00
Arteriosclerosis, cerebral	56	74.29	49,920.00
Arteriosclerosis, general	114	84.69	115,860.00
Neurocirculatory asthenia	1 625	24.39	475,620.00
Auricular fibrillation	34	81.62	33,300.00
Bradycardia	9	22.22	2,400.00
Cardiac arrhythmia, extrasystole	48	31.56	18,180.00
Cardiac arrhythmia, sinus arrhythmia	21	24.76	6,240.00
Cardiac disorder, functional	2	35.00	840.00
Cardiac hypertrophy	476	29.91	170,820.00
Cardiac hypertrophy and dilatation	13	58.08	9,060.00
Cardiac murmur, not organic	20	16.75	4,020.00
Degeneration and infiltration of heart	3	53.33	1,920.00
Dilatation, aortic arch	8	56.25	5,400.00
Dilatation, cardiac, acute	3	75.00	2,700.00
Dilatation, cardiac, chronic	14	51.79	8,700.00
Embolism, cerebral	7	62.86	5,280.00
Endocarditis, acute	1	50.00	600.00
Endocarditis, chronic	348	35.63	148,800.00
Endocarditis, septic	1	55.00	660.00
Functional heart disease	12	26.67	3,840.00
Heart-block	5	110.00	6,600.00
Hemorrhage, cerebral	15	74.00	13 320.00
Hypertension	196	32.14	75.600.00
Hypotension	9	32.22	3,480.00
Mediastinopericarditis	1	40.00	480.00
Myocarditis, acute	3	58.33	2,100.00
Myocarditis, chronic	2,639	40.26	1,275,060.00
Pericarditis, acute, fibrinous	1	50.00	600.00
Pericarditis, adhesive	21	46.90	11,820.00
Pericarditis, purulent	5	61.67	2,220.00
Pericarditis with effusion	11	77.73	10,260.00
Phlebitis, chronic	434	26.18	136,320.00
Phlebitis, suppurative, chronic	4	15.00	720.00
Phleboselerosis	1	100.00	1,200.00
Phlegmasia alba dolens	1	10.00	120.00
Pleuropericarditis	5	48.00	2,880.00
Tachycardia	490	20.62	121,260.00
Thrombophlebitis	99	31.77	37,740.00
Thrombosis	45	48.67	26,280.00
Valvular heart disease, aortic insufficiency	543	45.84	298,680.00
Valvular heart disease, aortic stenosis	148	49.36	87.660.00
Valvular heart disease, aortic and mitral	1,371	58.91	969 240.00
Valvular heart disease, mitral insufficiency	5,822	29.07	2,031,000.00
Valvular heart disease, mitral stenosis	1,314	37.87	597,120.00
Valvular heart disease, pulmonary lesion	8	26.88	2,580.00
Valvular heart disease, tricuspid lesion	3	75.00	2,700.00
Total	16,189	35.59	6,914,880.00
Grand total of active compensable beneficiaries	239,433	44.55	127.993,800.00

Per cent of total compensable beneficiaries receiving compensation for cardiovascular disease ---------- 6.76

Per cent of total annual outlay for compensation paid to beneficiaries for cardiovascular disease ---------- 5,40

Five thousand six hundred and forty-six, or 34.87 per cent, of the total number are on a permanent partial status. It was the opinion of the Veterans Bureau examiners that the cardiovascular disabilities of these cases were such as to constitute a permanent occupational handicap, but these men were not totally handicapped.

Seven hundred and seventy-eight, or 4.81 per cent, were placed on a temporary total compensation status. A great many of these beneficiaries were no doubt under hospitalization for the cardiovascular disease.

One thousand one hundred and four, or 6.82 per cent, of the total number of beneficiaries were on a permanent total status. In these cases it was thought by the Bureau physicians that the kind and character of the cardiovascular disabilities were such as to constitute a permanent total occupational handicap to the beneficiaries so grouped.

This, then, is the present cost to the United States of cardiovascular disease incurred by men and women who have been in the military service. A number of the beneficiaries with this class of disease have already died; an additional small number will be placed on the active compensation files of the Bureau from time to time after it is determined that their disabilities were incurred in or aggravated by military service in the World War.

TABLE XXII

SHOWING EXTENT OF DISABILITY, DEGREE OF IMPAIRMENT, AVERAGE MONTHLY COMPENSATION, AND THE ANNUAL COST OF COMPENSATION FOR 16,189 BENEFICIARIES WITH SERVICE-CONNECTED CARDIOVASCULAR DISEASE

EXTENT OF DISABILITY	DEGREE OF IMPAIRMENT (PER CENT)	NUMBER OF BENEFICIARIES	AVERAGE MONTHLY COMPENSATION (DOLLARS)	ANNUAL OUTLAY FOR COMPENSATION (DOLLARS)
Temporary Partial	10 – 19	2216	11.97	318,180.00
	20 – 29	2826	21.96	744,720.00
	30 – 39	1221	30.05	440,340.00
	40 – 49	692	38.23	317,460.00
	50 – 59	1072	47.35	609,120.00
	60 – 69	315	57.30	216,600.00
	70 – 79	281	68.83	232,080.00
	80 – 89	33	74.55	29,520.00
	90 – 99	5	87.00	5,220.00
	Total	8661	28.03	2,913,240 00
Permanent Partial	10 – 19	1871	12.65	284,100.00
	20 – 29	1931	22.69	525,720.00
	30 – 39	661	32.06	254,280.00
	40 – 49	347	41.31	172,020.00
	50 – 59	585	50.59	355,140.00
	60 – 69	121	61.86	89,820.00
	70 – 79	110	73.55	97,080.00
	80 – 89	19	83.16	18,960.00
	90 – 99	1	95.00	1,140.00
	Total	5646	26 54	1,798,260.00
Temporary Total		778	91.56	854,760.00
Permanent Total		1104	101.80	1,348,620.00
GRAND TOTAL		16189	35.59	6,914,880.00

The figures in Tables XXI and XXII stand out as excellent proof of the generosity of the people in providing amply for those who were disabled while in the service of their country.

SUMMARY AND CONCLUSIONS

1. a. There are 16,189 World War veterans at the present time with compensable cardiovascular disease. The living World War veterans number about 4,408,617. The incidence of this class of disease among the ex-service men is therefore approximately 3 per 1000. This is extremely low and the following reasons suggest themselves:

1. The average age of the World War veteran is approximately thirty-six years, and heart disease morbidity and mortality begin to ascend after the age of forty.

2. A large number of men with cardiovascular disease were kept out of the army by the draft boards, or, if accepted, were turned down at the various camps by the examining boards after it was ascertained that they had cardiovascular disease.

b. As the result of close contact in barracks during the war, infectious and contagious diseases were more prevalent among the military population than in civil life. This no doubt accounts for the high incidence of heart disease classified as the sequel of infectious diseases. This fact is one of the important observations in the study.

2. Of 736 Bureau patients under hospitalization for cardiovascular disease it was ascertained that there were a total number of 1123 lesions which were ascribed to 1761 etiological factors. It is supposed that the heart and blood vessels may be attacked by more than one disease, and each may leave an imprint of degenerative or inflammatory changes with resulting anatomical residua.

3. A consideration of the 1123 cardiovascular lesions found in 736 Bureau patients discloses the fact that the average number of lesions per patient is 1.53. The principal lesions are: mitral insufficiency, chronic fibrous myocarditis, hypertrophy of the heart, and aortic insufficiency.

4. a. In a study made of the specific cardiovascular lesions in a series of 736 Bureau patients it was noted that 453 had but one cardiovascular lesion; of the latter, the most common were mitral insufficiency, chronic myocarditis, and cardiac hypertrophy.

b. Two hundred and eighty-three patients had one principal and one associated cardiovascular lesion. The most frequent combination was mitral insufficiency with aortic insufficiency; the next most frequent combination was that of mitral insufficiency and hypertrophy of the heart; the next two most frequent combinations were those of mitral insufficiency and mitral stenosis, and cardiac hypertrophy and chronic myocarditis.

5. According to the etiological distribution of heart disease, 33.50 per cent of the cases were rheumatic; 30.04 per cent were sequelae of infectious diseases; 11.19 per cent were syphilitic; and 4.03 per cent were arteriosclerotic heart disease; the latter type is of interest in view of the fact that the average age of the living World War veteran is approximately thirty-six years.

6. A classification of 590 rheumatic factors according to the type of rheumatic infection causing heart lesions indicated that tonsillitis was the cause in 40.34 per cent of the heart lesions; acute rheumatic fever in 22.20 per cent; tooth and gum infections in 20.68 per cent; and the remaining rheumatic infections were such diseases as chorea, torticollis, pharyngitis, etc. These latter comprised 16.78 per cent of the total number of rheumatic infections.

7. In this study, heart lesions resulting from various infectious diseases were classified as sequelae of infectious diseases. It is noted that influenza, pneumonia, and tuberculosis were the principal diseases reported as the cause of this type of heart disease. The most common heart lesions occurring as the sequelae of infectious diseases were mitral insufficiency, chronic myocarditis, and hypertrophy of the heart.

8. a. A study of rheumatic heart lesions disclosed the fact that in a considerable number of cases it was impossible to ascertain the time elapsing between the occurrence of the rheumatic infection and the appearance of the heart lesion. However, in those cases in which it was possible to obtain the information, it was found that the time varied from one month in patients in whom the heart lesion developed coincidently with the rheumatic infection to twenty-five years in patients in whom the lesion was not recognized clinically until that length of time after the occurrence of the rheumatic infection. In the majority of cases the lesion developed within five years.

b. In the case of syphilitic heart disease the statistics compiled show that luetic cardiovascular disease appears in the majority of cases from five to ten years after the initial infection.

c. What was said of rheumatic heart disease may be applied to heart disease, the sequel of infectious diseases. The time elapsing between the occurrence of the infectious disease and the appearance of the lesion varied from one month in cases in which the lesion appeared coincidently with the infectious disease to twenty-five years in patients in whom the lesion was not noted until that length of time after the occurrence of the infectious disease. In the majority of cases, clinical evidence of heart disease was observed within five years after the infection.

d. It is assumed that the interval of time elapsing between the occurrence of the etiological disease and the appearance of the cardiac lesion is dependent upon several factors, such as:

1. The type of causative disease

2. Extent of degenerative or inflammatory changes and location of lesion or lesions

3. Functional activity to which the heart is subjected

9. a. A study of the duration of cardiovascular disease in 736 patients in the Bureau hospitals showed that in 245 cases it was impossible to determine the duration of the heart disease. In the remaining number, namely 491 cases, heart disease existed for periods varying from one to six months in 30 cases, to eight to nine years in 41 cases. In 3 cases the heart disease was congenital. The largest group of patients, 85 in number, gave a history of having had the cardiovascular disease from seven to eight years.

b. In the consideration of 255 of a series of 736 patients who were hospitalized primarily for some heart lesion, it was found that the most common lesions present were chronic myocarditis, combined aortic and mitral valvular disease, mitral insufficiency, aortic insufficiency, and mitral stenosis. Taking the whole group of 255 cases into consideration, the duration of the various lesions extended from the shortest period of one to six months to the longest period of eight to nine years.

c. In a study made of the duration of various cardiovascular lesions in 252 Bureau beneficiaries who died from cardiovascular disease, the average period from the date of diagnosis of the lesion to the date of death was as follows:

For acute dilatation, eight months; acute endocarditis, four years; chronic endocarditis, four years; cerebral hemorrhage, two years; acute myocarditis, four years; combined aortic and mitral disease, five years; chronic myocarditis, four years; and mitral insufficiency, three years.

The duration of the various heart lesions just enumerated is shorter than is the usual experience.

d. Consideration of the duration of heart disease* is fraught with a good deal of difficulty because:

1. Exact date of the inception of the heart lesion is not always known.

2. Wide range in the duration period of various heart lesions.

3. Duration period depends upon etiological type of lesion, as well as upon the site of the lesion.

*It is thought that the figures of Romberg, showing that approximately fifteen years is the duration of the average case of heart disease should not be stressed too much, nor should the latter figure be applied in an empirical manner, inasmuch as heart disease is so varied as to etiology and clinical course as to make the period of duration also varied. It is believed that in considering the period of duration of heart disease, cases should be treated according to the etiological types to which they belong.

4. Duration period depends upon the reaction of the heart muscle to the lesion present, i.e., upon the rapidity of compensation or decompensation.

10. a. A study of the functional capacity of 736 cardiovascular patients, grouped according to the classification of the American Heart Association, shows that 28.40 per cent were able to carry on their habitual physical activity; 29.62 per cent were able to carry on a slightly diminished activity; 19.29 per cent were able to carry on only a greatly diminished activity; 11.14 per cent were unable to carry on any physical activity and were confined to bed; and in 11.55 per cent of the patients the functional capacity was not noted.

b. It is thus seen that 60.05 per cent of the patients had heart lesions which were accompanied by a reduced functional activity. From an economic standpoint this is a serious handicap inasmuch as the earning capacity of these patients is greatly impaired.

c. It follows, therefore, that the Bureau has the same problems to solve in the case of the cardiovascular patients as it had in dealing with the tuberculous beneficiaries:

1. To rehabilitate these patients physically.

2. To restore them to a maximum economic state.

d. It is thought that the Bureau facilities in the hospitals and regional offices could be utilized not only to restore these cardiac patients to normal heart function, but that they could also be used in the instruction of the cardiac patients in various light occupational crafts for the purpose of increasing their work tolerance. The Bureau, through outside social agencies, should interest itself in securing suitable occupations for the claimants after they are dehospitalized. These patients should be urged to return periodically for cardiac examinations so that the effect of work on the circulatory system may be determined.

11. In a series of 145 cases in which the cardiovascular disease was attributed to infected tonsils, and in which tonsillectomy was done, it was found that in 18 cases the cardiovascular disease was benefited by the operation. The small number of cases showing improvement is possibly due to the fact that in the others additional foci of infection were present, and that the extirpation of the tonsils did not rid these patients of all the areas of infection.

12. a. In a study of 60 patients who died while under hospitalization for cardiovascular disease, it was found that the average number of lesions per patient was 1.6; 29.31 per cent of the lesions were of rheumatic origin; 12.07 per cent syphilitic; 18.10 per cent were sequelae of infectious disease, and in 31.90 per cent of the lesions the etiological factor could not be ascertained.

b. Of the total number of lesions (100) in the above 60 patients, the largest groups were those of chronic myocarditis, enlargement of the heart, aortic insufficiency, mitral insufficiency, and mitral stenosis.

c. Of the 60 patients referred to, 32, or 53 per cent, died from organic disease of the heart; 4 from acute endocarditis, and 13 from pulmonary tuberculosis. It is noted that in a number of the patients, even though cardiovascular disease was present, it was not necessarily the primary cause of death, but might have been a contributory factor.

13. The statistics of the United States Veterans Bureau indicate that the mortality from cardiovascular disease between 1922 and 1926, inclusive, has increased from year to year with the exception of the year 1924, when there was a decrease in the number of deaths. This increasing mortality due to cardiovascular disease is to be expected as the ex-service man approaches the age of forty.

b. Of the 939 deaths due to cardiovascular disease and occurring between 1922 and 1926, 661 were due to organic disease of the heart.

c. The Bureau statistics show that 46.6 per cent of the deaths due to cardiovascular disease and occurring between 1922 and 1926, took place within the age group from thirty to thirty-nine, and the percentage of deaths in the succeeding age groups became less. This may be explained by the fact that the majority of the ex-service men are within the age group thirty to thirty-nine, and the number becomes smaller in the succeeding age groups.

d. An analysis of the 661 deaths due to organic disease of the heart reveals the fact that the principal primary causes of death were chronic myocarditis, mitral insufficiency, combined aortic and mitral lesions, and acute cardiac dilatation. It is also noted that 524, or 79.2 per cent, of the 661 deaths occurred before the age of forty, and 137, or 20.8 per cent, occurred after that age. This is different from the customary experience in civil life and may be explained by the fact that the average age of the ex-service man is thirty-six yars and the age at death of most of these men falls within the age groups up to forty years.

14. a. In a study of 1,845 deaths of insured Bureau beneficiaries due to cardiovascular disease, mitral insufficiency was the cause 498 times; acute cardiac dilatation, 324 times; cerebral hemorrhage, 302 times; chronic myocarditis, 216 times; acute endocarditis, 179 times; combined aortic and mitral disease, 122 times; acute myocarditis, 104 times; and chronic endocarditis, 100 times.

b. Two hundred and fifty-two of the 1,845 deaths were studied to ascertain the average age at death. It was found that of the patients with acute cardiac dilatation, the average age was thirty-four years; acute endocarditis, thirty-two years; chronic endocarditis, thirty

years; cerebral hemorrhage, thirty-seven years; acute myocarditis, thirty-two years; chronic myocarditis, thirty-three years; combined aortic and mitral disease, thirty-four years; and mitral insufficiency, thirty-five years.

15. a. The active compensable beneficiaries of the Bureau number 239,433 of which number 16,189, or 6.76 per cent, are receiving compensation for cardiovascular disease.

b. The average monthly compensation of the beneficiaries with cardiovascular disease is $35.59; the average monthly compensation of all beneficiaries is $44.55.

c. The annual outlay of the government for the compensation of beneficiaries with cardiovascular diseases is $6,914,880.00; the total annual cost to the government of compensation for all disabilities is $127,993,800.00; the outlay for compensation for cardiovascular disease is 5.4 per cent of the total amount expended annually for the compensation of all classes of disabilities.

16. Of the total of 16,189 beneficiaries receiving compensation for cardiovascular diseases, it is found that 35.96 per cent are reported to have mitral insufficiency; 10.04 per cent neurocirculatory asthenia; 16.30 per cent chronic myocarditis; 3.35 per cent aortic insufficiency; 0.91 per cent aortic stenosis; 8.47 per cent aortic and mitral disease; and 8.12 per cent mitral stenosis.

17. Of the total number of beneficiaries receiving compensation for cardiovascular disease (16,189), it is noted that 53.50 per cent are on a temporary partial basis; 34.87 per cent are on a permanent partial status; 4.81 per cent are on a temporary total status; and 6.82 per cent are on a permanent total status.

In conclusion it is desired to acknowledge the cooperation in this study of Dr. A. E. Cohn of the Rockefeller Institute for Medical Research, and a member of the Research Group of the Medical Council of the U. S. Veterans Bureau.

It is also desired to acknowledge the encouragement of the Medical Director of the U. S. Veterans Bureau in this study; and the cooperation of the Evaluation Division, Central Office, and of the medical officers in the field; the latter were kind enough to furnish the data which form the basis of this paper. The assistance rendered by Miss Anne Bambery, Statistical Assistant in the Research Subdivision, in the preparation of the manuscript is greatly appreciated.

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THE ELIMINATION FROM THE ELECTROCARDIOGRAM OF THE EFFECTS OF ALTERNATING CURRENT OF POWER AND LIGHTING LINES*

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DURING the last two years I have been attempting to determine the cause of waves in the electrocardiogram which correspond in rate to that of the alternating current of the electric lighting and power lines in the vicinity of the electrocardiograph. During the investigation certain information has been obtained which may be of use to others confronted with the same difficulty.

In attempting to locate the cause of alternating waves in the electrocardiogram, the first thing to do is to determine whether the offending current is picked up by the electrocardiograph, by the leads from the patient to the electrocardiograph, or by the patient himself. To do this it is only necessary to take three test photographs: (1) with the patient in the circuit; (2) with the patient removed and the leads joined together, and (3) with the leads removed and a short wire connecting the lead terminals of the resistance-box. If it is found that current is induced on the connecting leads, the latter should be covered with some metallic tube, such as lead pipe. When the electrocardiograph is situated some distance from the patient, certain precautions must be taken in the installation of the lead wires. Specific information on this can be obtained from the manufacturers of electrocardiographs, the main precautions as given by them being: (1) The leads should be enclosed in a metallic sheath, such as lead, and this sheath grounded every fifty feet. (2) As far as possible paralleling the alternating current lines must be avoided; if this is impossible, as it is in a great many instances, there should be a distance of at least five feet between them. (3) All alternating lines should be crossed at right angles and in this case a distance of not less than eighteen inches should be between them.

If it is found that the interference is picked up by the electrocardiograph, it may be that by simply shifting the position of the instrument in the room, the difficulty can be overcome. Wires concealed in the walls and floor are often found to be the source of trouble. If the currents are creeping up from the floor to the instrument or the patient, dry insulators should be placed under each leg of the table on which the electrocardiograph is mounted, and the chair or bed on

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which the patient is placed. Grounding both the resistance-box and the galvanometer to a water pipe should be tried. Leaving the galvanometer ungrounded and at the same time grounding the resistance-box and vice versa in some cases gives results.

Parts of the instrument may, under certain conditions, produce waves in the electrocardiograph simulating alternating current. If the camera motor is suspected, this may be eliminated by disconnecting it from the camera and taking a picture while spinning the latter by hand. When the source of light is any but that of an ordinary direct current incandescent lamp, the possibility of the trouble originating at this source should be ruled out. An ordinary six volt automobile headlight supplied with current from a storage cell of the same voltage will serve as a very easy method to check this part of the instrument. The time marker may be easily excluded by taking a tracing with it disconnected.

During the investigation it was found that if the ends of two leads were connected together by a coil of wire similar to the coils used as resistances in the resistance-box, the tracing showed marked alternating current effects and, in fact, the vibrations of the string could be easily seen with the naked eye. This led me to believe that possibly currents were being induced in these coils during the taking of the electrocardiogram, thus causing the disturbance. A complete resistance-box was constructed in which all coils of wire were substituted by noninductive carbon resistances; this, however, did not reduce the alternating effect in the tracings. A simpler method of excluding the resistance-box is simply to take a tracing with the patient in the circuit without the resistance-box; the shadow of the string will be forced to one side of the field of light, but it is quite possible to take a tracing in this manner, especially if the skin current of the patient is small.

The effect of accumulation of static charges on the galvanometer may lead one to think that they are the cause of foreign waves in the electrocardiogram; these charges are evidenced by the string periodically going into violent vibrations the amplitude of which ranges from zero in which the string can be sharply focused to a maximum when it cannot be seen; one complete cycle usually extends over a period of about four seconds and is immediately followed by another. Allowing this charge to escape, either by grounding or by the operator touching the galvanometer, usually stops the vibration. It was found that this effect may be present to the most marked degree and at the same time a perfect electrocardiogram be obtained.

When it is found that the effect of the lighting and power circuits is present only when the patient is connected to the leads, it would be well first to try placing a large piece of galvanized iron under the bed or chair. A piece of thick linoleum or other insulating material

the size of the metal sheet should cover it; the best results may be obtained by grounding the iron to the lead covering of the leads, and the lead covering in turn should be grounded to the resistance-box. If the above efforts fail, then the room in which the patient is placed should be lined with some metallic substance, such as "gravel screen," the lining being compiete over the walls, ceiling, doors, and windows; the metal lining itself should be grounded. If the interference is not too great, placing a 4 M.F.D. condenser across the terminals of the string will give very good results and yet not modify the essential parts of the tracing to any great degree.[1] Valuable information on the protection of string galvanometer circuits against external electrical disturbances will be found in a paper by H. B. Williams.[2] If the electrical disturbance is of such a degree that none of the above measures eliminate from the tracing at all times the effects of alternating currents, it will be necessary to investigate the power and lighting lines in the vicinity of the instrument. In view of the fact that the electrocardiograph is disturbed only in certain localities, it occurred to me that the power lines in these districts must differ in some way from those in localities where there is no disturbance. It was found that the fault was not in the power and lighting lines themselves, but in certain motors, etc., receiving power from the same substation. These motors are supplied with 550 volts on a three phase system, which is not grounded. If a break occurs in the insulation of the windings of a motor, current passes from these windings into the frame and thus to the ground. This break in the insulation is evidenced at any point on the same line by a difference in potential between the other two phases and the ground. When first discovered, this difference of potential between one phase and the ground amounted to 540 volts. A recording voltmeter shows that the leak is not constant but varies from time to time, sometimes dropping to zero. Electrocardiograms taken when the voltmeter showed no leaks were free from alternating current effects, while those taken when the leak was large showed large waves corresponding in time to that of the local 25-cycle lighting and power circuit.

The solution of the problem is, therefore, in the repair or elimination of the offending electrical devices; these can best be traced out by the local electric lighting and power commission which will be glad to cooperate in the matter. It might be stated here that the first procedure is to conduct a thorough investigation in an attempt to locate the grounded machines and have them put in proper order or discarded; if it is impossible to find the trouble in this way, momentarily grounding the other phases will, for a time at least, remove the trouble and if often repeated will force the owner to discard the offending motor or other electrical device which is responsible for the leak.

SUMMARY

Information is presented which, it is believed, should prove of value in eliminating, from electrocardiograph tracings, the effects of disturbances from electric lighting and power circuits. The direct cause of the disturbance was found to consist in certain defective electric motors or similar devices receiving current from the same lines as the building in which the electrocardiograph is situated. The solution of the problem lies in the location and repair or elimination of the defective machines.

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Department of Clinical Reports

PLEURAL EFFUSION ASSOCIATED WITH CONGESTIVE HEART FAILURE LOCALIZED IN AN INTERLOBAR SPACE*

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STEWART† has recently reported pleural effusion associated with congestive heart failure, localized in an interlobar space. A rather careful survey of the literature reveals that the condition either is extremely rare or has not been reported. Hence the report of this case, which so closely parallels that of Stewart.

J. V. W., a male, fifty-one years old, traveling salesman, came under observation on May 28, 1928, complaining of dyspnea on the slightest exertion. Ten weeks before he had been recommended as a safe insurance risk by a competent examiner. A week later he noticed some difficulty in breathing. This had got steadily worse until on the day of examination, bending forward to tie his shoelace produced distinct shortness of breath.

The family history was unimportant and the past personal history negative. He had never had acute rheumatic fever, tonsillitis, chorea, or any of the streptococcic infections.

The present illness began nine weeks before he came for examination and was marked by constantly increasing dyspnea on exertion or haste, with slight cough, occasional spitting of blood, hoarseness, a sense of choking and gastrointestinal symptoms, especially the eructation of gas. He had had no previous medical attention.

The patient was an unusually robust man, 6 ft. 1 in. tall and weighed 235 pounds. There had been a loss of weight of ten pounds in the past eight weeks. He was markedly dyspneic on the slightest exertion. The skin was normally moist and there was no cyanosis. The frontal sinuses and antra transilluminated well. The pupils were regular and equal and reacted normally. There were several devitalized teeth. The tonsils had been removed. Examination revealed an enormously enlarged heart, but because of the thick chest wall it could not be definitely outlined by percussion. The heart sounds were remote and muffled and of a distinct tic-tac quality. There were no murmurs and no friction sounds. There were frequent premature contractions. The radial pulse rate was 108 with patient at complete rest. The systolic blood pressure was 180 mm. and the diastolic 120 mm. The breath sounds were not greatly altered except for scattered moist râles over the bases posteriorly. The liver was not enlarged, and there was no edema of the extremities. The prostate gland was normal.

An electrocardiogram showed an inverted T in the first and second leads and the QRS complex was slurred in all of the leads. There were frequent premature ventricular contractions. There was no axis deviation.

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†Stewart, Harold J.: Pleural Effusion Localized in an Interlobar Space, AM. HEART J. 4: 227, 1928.

The laboratory findings were as follows: *Blood:* Hemoglobin 87 per cent; red blood corpuscles 4,660,000; white blood corpuscles 6,700, with an increase in the polymorphonuclear neutrophiles to 83 per cent. *Urine:* Specific gravity 1.024 to 1.030; distinct trace of albumin; occasional finely granular casts and occasional pus cells. The nonprotein nitrogen was 60.6 mg. per 100 c.c. of blood and the uric acid was 4.6 mg. The Wassermann (blood) and Kahn tests were negative.

A flat plate of the chest taken at seven feet showed the heart enormously enlarged with the typical water bottle outline of pericardial effusion (no definite clinical signs of pericarditis were demonstrable). The cardiothoracic ratio was 75.7 per cent; the cardiac area (Bardeen) was 253 sq. cm. The striking finding in the

Fig. 1.—Localized pleural effusion and pericardial effusion.

roentgenogram was, however, a shadow seen in the right chest in the region of the interlobar space. (Fig. 1.) It was distinctly ovoid in shape and extended 8 cm. to the left of the right thoracic wall and was 5 cm. in width. The outline was perfectly regular and resembled, more than anything else, the shadow of a lemon. In density, the shadow was identical with that of the pericardial effusion.

The patient was put to bed and thoroughly digitalized. Clinical improvement was remarkably prompt and satisfactory. He stayed at rest for five weeks, but inasmuch as he was not in a hospital a further chest plate could not be obtained until July 12. (Fig. 2.) At that time the pericardial effusion had entirely disappeared, the cardiothoracic ratio had been reduced to 59 per cent, the area of the cardiac shadow was 198 sq. cm. (Bardeen) and the interlobar effusion had entirely disappeared. The cardiac rhythm was perfectly regular and compensation well

established. The blood pressure was 130/90 mm. No electrocardiogram was made.

The patient has continued to improve steadily and has resumed his former occupation. A seven foot plate made on January 5, 1929 was practically identical with that made on July 12, 1928, with no evidence whatsoever of the pleural effusion.

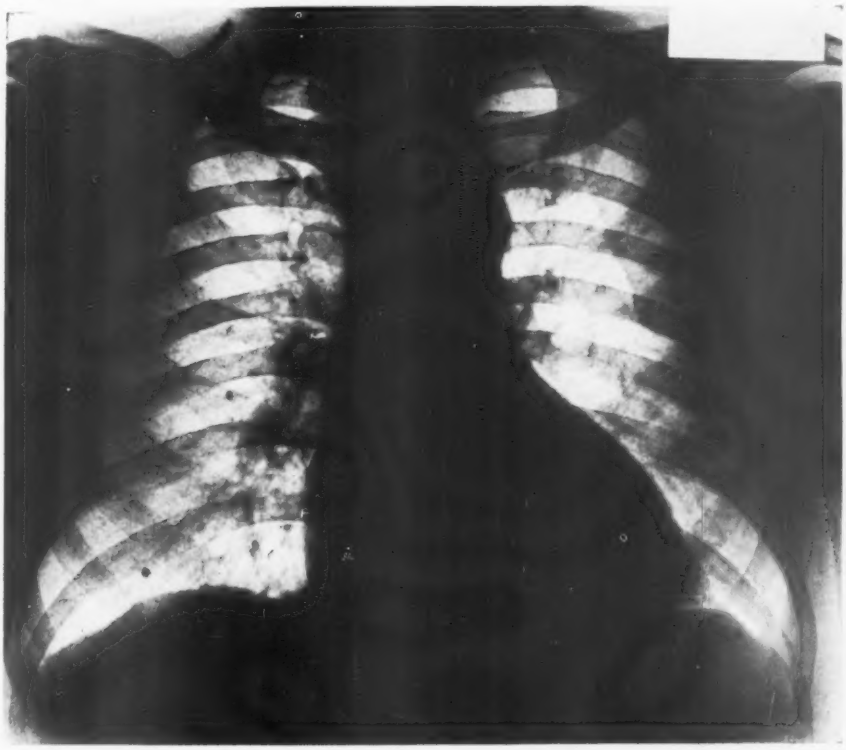

Fig. 2.—Note complete disappearance of both pleural and pericardial effusion.

The localization and character of the unusual shadow above described, together with its complete disappearance after the reestablishment of the cardiac compensation, the entire absence of any signs of neoplasm or empyema, justify, we think, the diagnosis of pleural effusion associated with congestive heart failure, localized in an interlobar space.

RETROPERITONEAL ABSCESS OF SPLENIC ORIGIN IN A CASE OF SUBACUTE BACTERIAL ENDOCARDITIS*

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THE following case is interesting because of the rare occurrence of suppurative processes following infarction of organs in subacute bacterial endocarditis and also because of the problem in diagnosis presented in such an instance.

J. K., a white woman, aged forty-two years, was admitted to the service of Dr. Hiram Loux of the Jefferson Hospital, July 19, 1928.

She complained of severe pain in the left upper quadrant of the abdomen. The family history was negative. The patient stated that she had been in good health until the onset of the present illness. She never had rheumatic fever, chorea, or tonsillitis. She had been pregnant eight times with normal labors. The present trouble began July 4, 1928, with severe pain in the left upper quadrant of the abdomen, fever, and urinary findings for which she was referred to the hospital with a note from the attending physician suggesting the diagnosis of pyelitis.

Cystoscopic examination revealed a normal bladder and normal ureteral orifices. Separate specimens of urine showed an insignificant number of white blood cells in each specimen. The patient seemed much improved after a week's rest and was discharged to a convalescent home on July 26, 1928.

Pain in the left upper quadrant recurred; she was readmitted to the hospital on September 4, 1928, and transferred to the medical ward on the service of Dr. Thomas McCrae, September 11, 1928.

Physical examination now showed a thin woman lying comfortably in bed. The mucous membranes were pale. The mouth and throat were in fair condition. The heart was enlarged to the left, almost to the anterior axillary line and a harsh blowing systolic murmur was heard at the apex. The pulmonary second sound was accentuated. The lungs were clear and resonant. The abdomen was distended, and the liver was enlarged to three fingers below the costal margin. There was a firm mass in the left upper quadrant of the abdomen which was tender at times. Neither physical nor roentgen-ray examination could establish that it was spleen, and yet it seemed continuous with splenic dullness. There was slight clubbing of the fingers. The temperature was very irregular with an occasional rise to 104°.

Urine examinations showed a trace to a cloud of albumin; a small number of pus cells, and occasional hyaline and granular casts, but no blood. Blood counts showed progressive secondary anemia with white blood cells varying between 6,000 and 10,000, the differential counts being unimportant. Numerous blood cultures were negative. Bacterial endocarditis was suspected, although it was felt that there might be additional trouble in the abdomen, such as perinephritic abscess. Pyelographic study of the left kidney showed apparently normal pelvis and calices, and additional roentgen-ray studies gave no evidence of perinephritic abscess. Nevertheless, the patient was operated upon October 10 under local anesthesia. A posterior incision exposed the left kidney but no abscess was found.

Roentgen-ray study on October 15 showed a mass in the left upper quadrant of the abdomen displacing the splenic flexure of the colon downward and inward.

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The lateral margin of the mass seemed to extend beyond the spine to the right, and the lower border was about the midpoint of the third lumbar vertebra. It seemed separate from the spleen which was somewhat enlarged.

On October 25 the blood culture showed a nonhemolytic streptococcus (not Streptococcus viridans) and this finding was confirmed on November 22. The patient never showed petechiae but shortly before death she complained of a very severe pain in the left wrist, and an area of bluish discoloration appeared on the ulnar side of the palmar surface extending into the palm of the hand. The temperature became very irregular with sharp peaks going to 104° and there were frequent sweats. Death occurred on December 7.

At autopsy (Dr. B. L. Crawford) the peritoneal cavity contained a slight excess of clear straw colored fluid. The abdominal organs occupied their usual positions. The spleen was enlarged and was densely adherent to the diaphragm and the lateral abdominal wall. There was a large retroperitoneal soft fluctuating mass on the left side of the abdomen which extended from the region of the spleen down to the brim of the pelvis. There was no evidence of inflammation of the general peritoneal cavity.

The heart was enlarged, weighing 420 grams; the muscle was soft and flabby. On the mitral valve and extending up on the wall of the auricle and down on the papillary muscles of the left ventricle were numerous long, pendulous, friable, reddish gray vegetations. Several were as much as 1¼ cm. in length. There was comparatively little thickening or deformity of the mitral valve leaflets. The aortic valve leaflets appeared normal. The coronary arteries were in good condition.

The spleen (430 grams) was enlarged and densely adherent to surrounding structures including the diaphragm, pancreas, and posterior abdominal wall. On releasing the adhesions a large pus pocket was found. This cavity was retroperitoneal and extended downward along the posterior abdominal wall to the brim of the pelvis. It contained about ½ liter of very thick greenish-yellow, foul-smelling pus. A large area of inflammation and necrosis was found in the diaphragm, and rupture had occurred into the left pleural cavity. The lung was adherent to the inflamed diaphragm.

There were several, circumscribed, soft grayish areas scattered throughout the spleen. On section some of these soft areas had partially necrotic centers and others had gone on to cavity formation. They had fibrous tissue walls and measured from 2 to 3 cm. in depth. Several of these reached the surface of the spleen and by rupturing, one such suppurative infarct must have been responsible for the retroperitoneal abscess.

The right kidney also contained a number of soft grayish infarcts.

Other organs did not show important changes.

Heart's blood culture yielded a nonhemolytic streptococcus and the same organism was recovered from the splenic abscess.

The significant findings therefore were: subacute bacterial endocarditis of the mitral orifice due to a nonhemolytic streptococcus (not viridans) and suppurative infarction of the spleen with retroperitoneal abscess formation and rupture of the left side of the diaphragm.

Department of Reviews and Abstracts

Selected Abstracts

Gladstone, Sidney A.: A Few Observations on the Haemodynamics of the Normal Circulation; and the Changes Which Occur in Aortic Insufficiency. Bull. Johns Hopkins Hosp. 44: 83, 1929.

The following paper presents a study of some of the phenomena associated with the physiology of a normal circulation and of the circulation as it is modified by various pathological processes in the light of principles of hydrodynamics.

The application of the hydrodynamic equation for the flow of water through pipes is made in an attempt to analyze certain problems in hemodynamics.

The forces concerned in cardiac contraction and the influence of respiratory movement are discussed. The fall of systolic pressure during inspiration is explained by the author as due to respiratory pressure changes in the mediastinum as the result of increased effort of respiratory movements. The change in blood pressure level during inspiration is just about equal to the changes of pressure which occur in the mediastinum during normal respiration.

Presents methods of calculating the work of the heart have underestimated the kinetic energy factor by failing to consider the systolic increase of velocity head imparted to the whole mass of arterial blood.

The general applicability of the principles of hydraulics to the problems of arterial flow is demonstrated by the author's ability to explain and correlate the findings of Dawson on the *Lateral Blood Pressures at Different Points of the Arterial Tree*. Brachial blood pressure is found to depend upon intra-aortic pressure head. Femoral blood pressure depends upon the sum of intra-aortic pressure head and velocity head, hence the so-called differential blood pressure.

The formation and propagation of the pulse are discussed. The present writer believes that the concepts of pressure waves have been applied erroneously in many problems of hemodynamics. For example, the nature of the pulse, the excess of femoral over brachial blood pressure, the difference between central and peripheral pulse, etc.

A well-developed case of aortic insufficiency is characterized by an exaggeration of the normal cyclic changes in pressure head, velocity head and total head of arterial blood. Evidence is offered for believing that the maximum velocity head especially increases above normal.

The present writer believes that the collapsing pulse is due to rapid egress of blood from the arterial system, resulting from the abnormally high systolic velocity head. A collapsing pulse diminishes the blood supply to the head and causes excessive mechanical strain of the arterial walls. An analysis is offered of the dynamics of regurgitation and the compensatory factors which tend to reduce the amount of blood regurgitation. The capillary pulse is believed to be a further consequence of the momentarily increased systolic velocity head and the exaggerated spurt-like character of arterial flood. The high femoral blood pressure in aortic insufficiency depends upon the high maximum velocity head of aortic and femoral blood. The brachial pressure may remain quite normal because the brachial and subclavian arteries arise at right angles from

the arch of the aorta and act as piezometer tubes, their flowing depending entirely upon pressure head and not being directly influenced by changes of intra-aortic velocity head.

The difference in mass of blood is only of secondary importance and may exaggerate the effect of water hammer. It is considered unnecessary to postulate an unequal distribution of pressure waves reflected from the periphery or even their existence in attempting to explain the excess of femoral over brachial blood pressure.

Gladstone, Sidney A.: Concerning the Mechanism of Production of the Korotkoff Sounds and Their Significance in Blood Pressure Determinations. Bull. Johns Hopkins Hosp. **44**: 122, 1929.

The present writer offers an explanation of the method of production of the Korotkoff sounds based on an analysis of the various factors concerning their change during decompression of the cuff and their effect upon each other.

The sounds are produced by sudden distention of an artery, the walls of which were previously relaxed because the pressure of the surrounding pneumatic cuff opposes the diastolic intra-arterial pressure and keeps the artery undistended during diastole.

The appearance of the first sound is a measure not of systolic pressure head but rather of the sum of pressure head, velocity head and such increment as may be due to water hammer.

The dulling of the sound at the beginning of the fourth phase probably occurs just below the intra-arterial diastolic pressure head.

During the second phase the arterial sound weakens or disappears and is supplemented or replaced by a murmur. In such cases of hypertension or aortic stenosis, where the rate of arterial blood flow is not rapid enough to produce a murmur during the second phase, there is produced an auscultatory gap, since there is nothing to supplement or replace the arterial sound which during that phase weakens or disappears.

Lundy, Clayton J., and Woodruff, Lewis W.: Experimental Heart Block. Arch. Int. Med. **43**: 184, 1929.

In the course of experimental work on the heart a new method was devised for increasing the intra-auricular and intraventricular pressures of the intact dog. This was done by tying a balloon on the tip of a properly bent glass rod and inserting the rod into the right auricle or ventricle by way of the right jugular and innominate veins. This apparatus was connected with a mercury manometer and a pressure bottle which permitted raising the intra-auricular pressure to any desired amount.

With an intra-auricular pressure of from 15 to 60 mm. of mercury, there was gradual lengthening of the P-R interval from 0.08 second to 0.2 second, a period of 2:1 block, establishment of an independent ventricular rhythm and finally complete suppression of the P-wave.

After release of the pressure in the balloon, there was a gradual return to the control type of sinus rhythm with normal P-R intervals.

Master, Arthur M., and Oppenheimer, Enid Tribe: A Simple Exercise Tolerance Test for Circulatory Efficiency With Stand Tables for Normal Individuals. Am. J. M. Sc. **177**: 223, 1929.

The test here described utilizes habitual muscular movement causing a minimum of excitement and no dizziness or vertigo. It consists in ascending and

descending two steps, each 9 inches high. The number of foot pounds of work performed is easily calculated.

The advantages of a quantitative method for an exercise tolerance test are described, and its value in diagnosis is emphasized.

The criterion for the satisfactory performance of the test is a return of systolic blood pressure and of pulse rate to the pre-exercise level within two minutes after the end of exercise. Tables have been constructed giving foot pounds of work per minute performed by normal individuals with variation for sex, age, height and weight.

Curves of food pounds of work per minute plotted against age show that until about puberty girls perform more efficiently than boys, but after thirteen years males do better than females. The sharpest rise in the curve occurs between ten and twenty years of age. Males reach a maximum of about 3795 foot pounds of work at about twenty-six to twenty-nine years and women a maximum of 2950 at about twenty-four years. After this optimum of ages a gradual but steady decline occurs.

Exercise tolerance varies directly with body weight up to the optimum weight of 160 to 165 pounds for men and 135 to 145 pounds for women. Beyond these weights the exercise tolerance falls.

The relation of work capacity to height is a direct one, the taller the individual the greater is his exercise tolerance.

The practical importance of the procedure is that the patient can be tested to determine: first, whether his exercise tolerance is within normal limits; second, what his actual maximal tolerance is in foot pounds.

The test is helpful in the diagnosis and grading of circulatory efficiency and for tracing the changes arising therefrom during the progress of an organic condition of the heart or during recovery. It may also be used in giving advice as to sports and games.

Farrell, John T., Jr., Langan, Paul C., and Gordon, Burgess: A Roentgen Ray Study of a Group of Long-Distance Runners. Am. J. M. Sc. 177: 324, 1929.

The present roentgenographic study was undertaken to determine the effect of long-distance running in 23 athletes. In a race from Los Angeles to New York these runners averaged 41 miles daily for 84 consecutive days. The study was made three days after the end of the race. They would indicate that when changes in the bones or sudden death are caused by athletic training or competition that the individuals were unqualified physically for strenuous effort, and exercise merely exerted a precipitating influence. In the teleroentgenograms the greatest transverse diameter of the heart was measured for comparison with the tables of Bardeen. The predicted diameter for each runner was computed for height and weight and an average obtained which was considered the normal diameter for the given individual.

The lungs were found to be essentially normal. The bones, except for certain osteoarthritis changes, were normal. The blood vessels were visualized only in the older runners.

In 13 runners the hearts appeared to be definitely smaller than normal; in 5 runners the hearts were within normal limits, and in five instances the hearts according to the predicted diameters showed an increase in size. According to the so-called cardiothoracic ratio, only one heart was increased in size.

The data as a whole suggest that the immediate effects of long distance running are inconsequential, since all the changes noted may be found in individuals of similar ages without symptoms.

Mond, Herman, and Oppenheimer, Enid Tribe: Gallop Rhythm in Hypertention. Arch. Int. Med. 43: 166, 1929.

In this paper the authors have attempted to elucidate the dynamic factors connected with the production of gallop rhythm by means of graphic methods giving exact records of the sounds and their relationship to the electrical and mechanical phenomena of the heart cycle. They have studied only patients presenting the classic syndrome of Potain, that is gallop rhythm in cases of hypertension with more or less marked cardiac insufficiency. By means of simultaneous electrocardiograms, phonocardiograms and apex impulse tracings, they have been able to ascertain the exact time relationships of the extra sounds to other events of the cardiac cycle and have considered which of such events may have a casual connection with the sound. They have found in agreement with all previous observations that in true gallop rhythm the extra sound occurs during ventricular diastole. The curves show that this extra sound on the phonocardiogram always occurs at the same time as the latter half of the P-wave and simultaneously with a large distinct wave of the apex beat and the A-wave of the venous pulse. This would, therefore, point to the auricular contraction as the determining factor.

As far as is shown by the records it would appear that the distinction between protodiastolic and presystolic gallop is not of great significance. Granting that in these cases gallop sound and auricular contraction are interrelated, it will depend entirely on the relative position of auricular contraction between the preceding second and the subsequent first sound whether the gallop is protodiastolic or presystolic. The factors determining this are, therefore, the rate of the heartbeat, on the one hand, and the length of the auriculoventricular conduction interval on the other.

They discuss the differentiation that must be made between gallop rhythm and the physiologic third sound and the reduplication of the second sound in mitral stenosis.

From their studies the authors believe that the mechanism of gallop rhythm is explained, as it was originally by Potain, as due to a sudden distention of the rigid fibrosed muscle wall of the ventricle by the inflowing blood from the auricle. In Potain's description, however, the rôle of the auricular contraction is neglected. The authors, however, believe that the auricle contracting plays an important part in the production of this sound. They proceed to discuss two questions: one, why is auricular contraction not heard under normal conditions; two, what are the conditions that make auricular systole an audible event? They believe that the auricle contracting is not heard naturally because there are factors causing the sound vibrations to be damped. Due to this fusion of the ventricular sound, the vibrations produced by the auricle contracting fall beyond the level of audibility. In the normal heart the ventricle is completely relaxed when auricular contraction occurs, its walls forming a soft, flexible mass which yields readily to the slightest pressure of the inflowing blood. In this phase auricle and ventricle form one large cavity, whereof the soft ventricular portion not only fails to participate in the vibrations of the auricle but also acts as a damper that silences whatever vibrations may have been started by the sudden contraction of the auricle muscle fibers. The amount of damping is clearly proportional to this yielding capacity of the normal ventricle muscle in diastole.

With rising tension of the ventricular wall the damping influence must be diminished and the chances of audibility improved. In the cases under dis-

cussion it is probable that factors are present in the ventricular muscle which make it favorable for the appearance of the sounds of auricular contraction.

The authors assume in explanation that the ventricular wall is no longer in a state of complete relaxation in diastole but now offers a marked resistance to the entrance of blood during auricular systole. The pressure wave produced by the contracting auricle meets with a ventricular wall which no longer yields passively to every impulse. The pressure is raised suddenly in the large common cavity comprising both auricle and ventricle, and the whole of its surrounding wall is thrown into transverse vibrations. These transmitted to the wall of the chest are heard as the familiar third sound of gallop rhythm. As has often been stated, it is best heard near the apex which is that portion of the vibrating muscle mass closest to the wall of the chest. In this conception the extra sound is a combined auricular and ventricular effect, and the participation of the ventricle explains why a sound, auricular in origin, should be so well perceived over the ventricle.

Symposium on the Aspects of Chronic Heart Disease. New England J. Med. 200: 1, 1929. **1: Experience with Chronic Cardiac Patients at The Robert B. Brigham's Hospital.** Spear, Louis M.

The following points were emphasized as the result of this experience: (a) patients with endocarditis must be kept quiet as long as there is evidence of cardiac irritability; (b) there is a definite tendency to recurrence of cardiac infection under the best of control conditions; (c) the necessity for long rest in a hospital for the majority of cases, especially for those where home conditions are not suitable, to provide adequate rest; (d) the difficulty of control in cases that have been discharged to the Home Service is pointed out.

2: Obtaining Occupation for Adult Males With Heart Disease. Raymond, Howard C.

The usefulness of the classification of heart patients adopted by the New York Heart Committee has been proved in the placement of patients. It is pointed out that the doctor frequently may state what the patient cannot do, whereas the idea uppermost in the worker's mind is what he can do. Intimate relationship between the placement worker, the hospital social service worker and the employer is necessary. Various types of work suitable for male cardiac cases are enumerated.

3: The Placement of Women With a Heart Handicap. Fletcher, Gertrude L.

The author feels that the principles in placing women are the same as those in placing men. There is, however, one great advantage in placing women in the fact that many of the jobs calling for unskilled women are light, whereas those for unskilled men are heavy. On the other hand, there are many more places for unskilled men than for unskilled women.

4: Home Adjustments in Chronic Heart Disease. Upton, Nathalie B.

The adjustment of home problems stands out as one of the most challenging as well as one of the most hopeful problems presented. The author discusses the various phases in the home that require adjustment in order for the patient properly to meet his daily needs and life. How these home adjustments are to be made is a matter of technic which varies with the individual worker, the individual patient and the problem.

5: **Instruction in the Home for Children Handicapped by Heart Disease.** Terry Edith M.

The various questions arising in the care of young children with heart disease, as well as those during adolescent life, are presented. The need for supervision in school, in the home, in outside activities and preparation for vocational occupation are pointed out.

6: **Vocational Guidance for Children With Heart Disease.** Ginn, Susan J.

This particular phase of adapting children for future occupation is discussed. Interesting examples are cited where material benefit resulted to the patient from this phase of social work.

Heimann, H. L., Stracham, A. S., and Heyman, S. C.: Cardiac Disease Among South African Non-Europeans. Brit. Med. J. 344, Feb. 23, 1929.

The authors have analyzed the notes of all cases of heart disease among non-European patients of the Johannesburg General Hospital from March, 1924, to March, 1928. Although the cases are few in number, they serve as a useful guide for future investigation and represent one of the first reports of this disease among a native African population. The persons included in this study were those working in large towns and cities and therefore in contact with European population. Also they represent the more severe types of heart disase, since such people do not apply for admission until they are in the last stages of this disease. In all, there are notes on 153 cases of which 120 were Kaffirs, 31 were Eurafrican and 2 were Hottentots. Eighty-one died in the hospital. Approximately two-fifths of the patients were of rheumatic type. Of these 64 patients only about one-sixth gave a rheumatic history. There were 39 cases with syphilitic infection; 25 cases had degenerative lesions; 14 cases of bacterial endocarditis, and there were 8 persons with pericarditic lesions. The changes found at autopsy were similar to those described among European and American population.

The authors conclude from this study that there is very little divergence between the etiology, pathology and clinical signs of heart disease in Europeans and African non-Europeans.

Some important etiological factor in the causation of rheumatic fever might be elucidated by comparing the Kaffirs living altogether in the native state with the Kaffirs in the towns. The medical history of the Kaffirs before the influence of European civilization affected him is unfortunately not to be ascertained. The climate of South Africa, speaking generally, is one of sunshine and is hot and dry. It seems that climatic conditions as an etiological factor in the causation of rheumatism must be discountenanced here. Nasopharyngeal infections or bad teeth cannot be dismissed as factors, as they commonly occur in hospital patients. Their evidence in this series has not been determined. The dietary factor is a very variable one in the series. It is difficult to obtain exact knowledge but the Kaffir, as a rule, takes a diet of greater carbohydrate content and less fat and vitamine constituent than the European.

In regard to nonrheumatic cases little can be added to the facts already known.

White, Paul D., and Hahn, Richard G.: The Symptom of Sighing in Cardiovascular Diagnosis. Am. J. M. Sc. **177**: 179, 1929.

The study of sighing has included observation and inquiry of its frequency in 650 cases mostly adults of whom 400 were normal controls, nearly all young men and women. One hundred were patients of organic heart disease who were

not especially nervous or tired. One hundred were patients of nervous fatigue or effort syndrome without heart disease, and 50 were patients with both organic heart disease and effort syndrome. Among the normal control persons sighing is a common finding but of frequent recurrence in only 19 persons. It was especially common as a frequent event in young women in the third decade of life, and it is generally recognized that it is this sex at this age that shows the greatest degree of nervous instability.

Frequent sighing is relatively rare in heart disease with or without congestive failure if there is no effort syndrome or marked nervousness.

In effort syndrome without heart disease excessive sighing is very frequent (80 per cent of 100 cases). When effort syndrome or marked nervousness is combined with organic heart disease, sighing is common. It is evident from the data that in these cases the sighing comes from the nervous state and not from heart disease.

Levy, Robert L., and Turner, Kenneth B.: Impaired Auriculoventricular Conduction in Rheumatic Fever. Arch. Int. Med. 43: 267, 1929.

In following a series of cases of rheumatic fever in which frequent electrocardiograms were made, the diagnostic importance of disturbances in auriculoventricular conduction became apparent. The records of all rheumatic patients in the course of nine years showing prolonged conduction or heart-block were critically analyzed in this study. For purposes of comparison all other cases showing disturbance in auriculoventricular conduction were similarly reviewed. A total of 145 cases was found. Of these 127 showed a prolonged P-R interval. More than one-half of these cases were instances of rheumatic fever. This prolongation of conduction time is by far the most frequent disturbance in this disease.

The authors have also studied the question of the involvement of the myocardium in cases of rheumatic fever. In their study they have found that of 403 patients, 112 (27.8 per cent) showed a long P-R interval.

A prolonged interval or heart-block occurring in a person under the age of thirty-five years who is not syphilitic and has not taken digitalis affords presumptive evidence of the presence of rheumatic carditis. This sign is often useful in establishing the rheumatic nature of a cardiac disturbance in the absence of other criteria. In 3 cases of acute tonsillitis, they found that there was definite prolongation of conduction time. They point out that in such cases there may be myocardial involvement of a rheumatic nature not recognized otherwise.

Prolongation of auriculoventricular conduction may persist and give evidence of myocardial lesions in rheumatic fever long after the other clinical manifestations of the disease have subsided.

Adams, S. Franklin: A Study of the Blood Pressure of Patients with Diabetes Mellitus. Am. J. M. Sc. **177:** 195, 1929.

In this study the author has investigated 1001 diabetic patients in the Mayo Clinic. Arbitrary standards were chosen for high pressure and low pressure groups. The patient of any age was considered to have a high blood pressure if the systolic pressure was more than 150 mm. or if the diastolic pressure was more than 100 mm. In this group 16.2 per cent of the male diabetic patients, showed such an elevation of pressure and 26.7 per cent of the female diabetic patients had a similar elevation. The patient of any age was recorded as having low blood pressure if the systolic pressure was less than 110 mm. or the diastolic pressure was less than 70 mm. Of male diabetic patients 15.6 per cent had a systolic blood pressure lower

than 110 mm., and 19 per cent of diabetic women had systolic blood pressures less than 110 mm. These data like those of the high blood pressure group are averages for all ages. The final "weighted" averages of the group do not disclose any significant difference between the blood pressure of diabetic patients and that of normal persons.

The author concludes that there is no clear evidence that diabetes of itself promotes hypertension. If the blood pressure of diabetic patients is elevated, it suggests the presence of some associated abnormality which of itself is responsible for the hypertension.

Amberg, Samuel: Hypertension in the Young. Am. J. Dis. Child. **37:** 335, 1929.

Twenty-five children with increased blood pressure and ranging from the age of six to sixteen years were examined and treated at the Mayo Clinic during the period of about eight years. A study of this group of cases forms the basis of this report.

The author divides the cases into the following groups: (1) hypertension associated with coaretation of the aorta; in these patients the elevation in blood pressure is confined to the upper part of the body, there is relatively low diastolic pressure, and there were pulsating superficial areas on the thorax; (2) hypertension as the consequence of an organic cerebral lesion; (3) hypertension perhaps in the form of so-called essential hypertension; (4) hypertension in the form of a syndrome of malignant hypertension, as described by Keith, Wegener and Kernohan with changes mainly in the small arteries and arterioles. These cases are characterized chiefly by persistent hypertension, absence of higher grades of anemia, adequate renal function at a time when striking lesions of the eyegrounds are visible and rapid fatal ending. The most important and constant feature is the extreme diffuseness of the histologic changes in the arteries and arterioles. These changes show a thickened muscular coat and hypertrophy of the lamina elastica which is split in places. (5) Hypertension associated with cardiac decompensation; (6) hypertension intimately associated with disease of the kidney. This whole group is not composed of homogeneous material, but no further attempt at a subdivision is made.

Eggleston, Cary: The Persistence of a Mitral Stenotic Murmur in the Presence of Auricular Fibrillation. Am. J. M. Sc. **177:** 153, 1929.

It is the purpose of this communication to show that it is not uncommon to encounter patients with rheumatic mitral disease and fibrillation of the auricles in whom a mitral murmur is clearly heard to continue right up to the first sound of the heart. In a considerable portion of such cases this murmur sounds definitely crescendo at its termination in presystole and is, therefore, identical with that murmur which is regarded as most characteristic of mitral stenosis when the regular sinus rhythm is present.

The author believes that this apparent paradox is due to an auditory misinterpretation produced by the shortening of diastole and the accentuation of the first heart sound.

He states that two definite and distinct murmurs occur with stenosis of the mitral valve. The one is an isolated short murmur occurring synchronously with contraction of the auricles. It may be soft, blowing and rather indistinct, or, less commonly, it may be rough and rumbling. It usually gives the impression to the ear of being crescendo. This murmur is correctly attributed to the influence of auricular systole and should properly be called the auriculosystolic murmur.

The second murmur is not limited to presystole but begins at or before mid-diastole and continues through to end with the first sound in the heart. It is almost always coarse and rumbling, and it frequently seems to increase in intensity in presystole so that to the ear at least it appears to be clearly crescendo at its ending. This is the murmur which is most commonly present, and as in the case of auriculosystolic murmur it too is very commonly called a presystolic murmur. Since this murmur occupies a considerable portion of diastole and is never confined to presystole, it is much better to speak of it as the diastolic murmur of mitral stenosis.

In the course of this investigation in a large number of patients who have mitral stenosis and auricular fibrillation, the author has never heard the isolated auriculosystolic murmur. The second type of murmur which begins at an earlier stage of diastole is the one which may remain after the auricles have passed into fibrillation. Its persistence has now been observed in more than 40 patients. All of the patients were suffering from rheumatic heart disease, and in all the evidences of mitral stenosis were such that this clinical diagnosis could not have been called into question. The presence of auricular fibrillation was established by electrocardiography. Several of the patients who died were proved at autopsy to have had advanced degrees of mitral stenosis.

The mechanism by which these murmurs are produced is that of the formation of a jet. Narrowing of the mitral orifice yields almost ideal conditions for the production of a rapidly flowing jet of blood as the stream enters the ventricle. When the stenosis is marked, the jet should occur throughout the greater part of ventricular diastole. In very pronounced stenosis it should persist until the intraventricular pressure begins to be raised to the level of that in the distended auricles by the onset of ventricular systole. Higher grades of stenosis are always accentuated by diastolic murmurs, and the duration of the murmur depends upon the degree of stenosis, the tighter the stenosis, the longer the murmur.

The author believes that the murmur under consideration never ends with a crescendo reinforcement in the presence of auricular fibrillation. The apparent crescendo ending is an auditory illusion due to the abrupt termination of the rough murmur by the loud sharply accentuated first sound which has the pitch that is not widely different from that of the murmur itself.

Palmer, Robert S., and White, Paul D.: The Clinical Significance of Cardiac Asthma. J. A. M. A. 92: 431, 1929.

In a group of 250 patients with cardiac asthma discovered in the past few years among 3100 private and hospital patients with organic heart disease, 180 were males, 70 females, and all except 14 were over forty years. The grave prognostic significance of the condition is shown by the fact that 170 of the 250 patients were known to have died with an average duration of life of 1.4 years after the first attack of cardiac asthma. The largest number of cases, 187, was found in a group of patients with coronary disease, hypertension, or both, but the highest relative incidence occurred in syphilitic heart disease and in chronic nephritis. Left ventricular failure due to any one or a combination of several factors appears responsible for cardiac asthma, but the exact mechanism is not clear. The frequency, duration and severity of the attacks alter the prognosis appreciably only when of an extreme degree. The coincidence of poor heart sounds, gallop rhythm and pulsus alternans indicated as a rule a very short life. Aortic regurgitation usually of syphilitic origin was the only common valve defect.

In therapy, digitalis and rest were generally effective in reducing the number of attacks and apparently prolonging life; for treatment of the acute attacks

nitrites and alcohol were sometimes helpful, while morphine was of the greatest value.

The mechanism of cardiac asthma as studied in this present group may be explained as follows. As a result of left ventricular strain and failure, with increased flood flow, a stasis of blood in the pulmonary circulation develops, the right ventricle sending on too much blood for the left ventricle to take care of. Gradually this accumulation of blood in the pulmonary circuit increases until by reflex stimulation the attack of asthmatic breathing is induced and the patient is awakened. By assuming the upright position, the stasis and also the strain on the left ventricle are reduced and recovery takes place.

White, Paul D., and Sprague, Howard B.: The Tetralogy of Fallot. J. A. M. A. **92:** 787, 1929.

The tetralogy of Fallot consists of the most common grouping of congenital cardiac defects found in adults, namely, (1) pulmonic or infundibular stenosis; (2) interventricular septal defect; (3) dextroposition of the aorta, so that it overrides the septal defect and thus receives blood directly from the right as well as from the left ventricle; and (4) marked hypertrophy of the right ventricle. Clubbing of the fingers and cyanosis are invariably present.

The authors report the case of a notable musician, Henry F. Gilbert, who for nearly sixty years of his life suffered from congential heart disease with cyanosis and clubbing of the fingers. The case is of interest first because he survived to his sixtieth year surpassing in age all patients previously reported by more than 23 years, and secondly and most significant because he made a great success of his crippled life, establishing himself in his musical profession as one of the greatest of American composers and as a pioneer of native American music. The history, accomplishments, physical examination, laboratory data and the conditions found post-mortem in this remarkable man are recounted.

The diagnosis of the cardiac defects was correctly made a year before his death. Fallot had demonstrated that this was possible forty years ago.

The authors report the case, believing that the account of this man's life may bring hope and inspiration to other victims of heart disease.

Richardson, Edward P., and White, Paul D.: Sympathectomy in the Treatment of Angina Pectoris. Am. J. M. Sc. **177:** 161, 1929.

Eight patients with angina pectoris were operated upon at the Massachusetts General Hospital between August, 1923, and December, 1926. Since February, 1927, paravertebral injections of 85 per cent alcohol according to the method of Swetlow have been used instead of operation.

Among eight patients having eleven operations, there were two operative deaths. A lasting benefit followed operation in four cases and was directly attributable to it in three. On considering these results early in 1927 the authors were on the whole not greatly encouraged in continuing with sympathectomy of angina pectoris. As an alternative means of treatment it was decided to try the effects of paravertebral injection of alcohol.

Altogether eight patients have been treated by this means. These cases as a whole were comparable in seriousness to those treated by operation. The patients were either incapacitated or bedridden on account of the severity and frequency of their attacks. Three of them had coronary thrombosis and so would not have been suitable risks for operation; they survived the paravertebral alcohol injection without difficulty.

Symptomatic relief has more constantly been secured by paravertebral injections than by operation. While there was a tendency for pain to recur in

one or two cases, the relief generally lasted as long as the patients have been followed, up to eighteen months in one case.

The authors believe that treatment is justifiable in carefully selected patients who do not respond to other forms of treatment. They summarize the selection of cases as follows: paravertebral alcohol injection, generally of the left upper five thoracic nerve roots may be recommended in the treatment of obstinate angina pectoris which persists in spite of ample medical measures and which renders work impossible and life miserable. With these indications there seem to be no contraindications. The injections may be made even after coronary thrombosis and doubtless are much less risk than is cervical sympathectomy. It is quite possible that failure of a paravertebral alcohol injection may justify operative procedures, but their experience to date would indicate that the therapy of first choice for angina pectoris when medical measures have failed is the injection method.

Gilbert, N. C., and Kerr, John Austin: Clinical Results in Treatment of Angina Pectoris With the Purine-Base Diuretics. J. A. M. A. **92:** 201, 1929.

All the drugs of this series have been used for the relief of anginal pains with ambulatory patients in the out-patient clinic discharged from the medical wards of St. Luke's Hospital, Chicago, since 1917. The authors describe the detailed technic of observing patients and the methods of administering these drugs. Their results are summarized in tables and are based on a total of 86 patients. They believe that the purine-base diuretics are of very definite value to many patients suffering from angina pectoris and should at least be given a fair trial in such cases. One preparation may be of greater value than another preparation very similar chemically, and the exact choice will depend on the individual study of each patient.

The authors believe from the study of these patients that the phenomena of angina pectoris can best be explained by a blood flow in the coronary vessels inadequate to the needs of the heart muscle at the moment. The purine-base diuretics are drugs capable of producing increased coronary flow. Beneficial results could then be expected when an increased coronary flow was not precluded by advanced anatomic changes in the vessels.

Eddy, Nathan B., and Hatcher, Robert A.: The Seat of the Emetic Action of the Digitalis Bodies. J. Pharmacol. & Exper. Therap. **33:** 295, 1928.

The present investigation shows that nicotine abolishes the emetic action of pure digitalis principles which are in common use and also that of many of the crude drugs of this group.

A severely toxic dose of nicotine may be administered to a cat every half hour during many hours, showing that the drug is eliminated rapidly, but the action of nicotine whereby it abolishes the emetic action of digitalis bodies persists for three hours or more.

Dresbach, Melvin, and Waddell, Kenneth C.: The Emetic Action of Digitalis Bodies and Strophanthidin in Cats With Denervated Hearts. J. Pharmacol. & Exper. Therap. **34:** 43, 1928.

The authors have examined experimentally the hypothesis that the origin of vomiting induced by digitalis bodies arises in the cardiac muscle by denervating the heart of the cat in survival and acute experimetns.

It was shown that with the heart thoroughly denervated in survival experiments digitalis bodies or strophanthidin or both induce emesis with certainty.

It was further shown in acute experiments and in which the time for recovery from operative effects was greatly reduced that the animals failed to vomit in the greater number of cases. The conditions of the experiments in these cases strongly suggest, in the light of those of the longer method, that it is not safe to use the shorter method in the attempt to locate the seat of action in digitalis emesis.

The conclusion drawn is that in experimental animals, the heart cannot be said to be the sole seat of action of digitalis emesis; whether or not the same thing may be true of this emesis in intact animals is not yet known with certainty.

The possibility of strophanthidin acting on the peritoneum, as well as on the heart, to induce emesis is examined and seen to be not well founded, though such a possibility has not been disproved.

Bond, W. R., and Gray, E. W.: The Supposed Influence of Polarized Light on the Deterioration of Digitalis. J. Pharmacol. & Exper. Therap. 32: 351, 1928.

Studies have been made in connection with the deterioration of digitalis with age, particular attention being paid to the conditions under which the preparations have been stored. In view of the fact that certain tinctures exposed to daylight and artificial light for a period of time ranging from one to five years did not show any appreciable change in toxicity when assayed by the cat method and with the support of experimental evidence on tinctures radiated with polarized light of considerable intensity, the authors believe that the potency of digitalis preparations, as judged by the cat method of assay, is not lowered by exposure either to ordinary or to polarized light.

Gold, Harry, and DeGraff, Arthur C.: Studies on Digitalis in Ambulatory Cardiac Patients. II. The Elimination of Digitalis in Man. J. Clin. Investigation 6: 613, 1929.

The authors have been studying the various phases of the question of the elimination of digitalis and the duration of its effects in man. The studies have been carried out upon ambulatory cardiac patients with auricular fibrillation because in these the onset and disappearance of digitalis effects could be established with greater precision. The ventricular rate was used to indicate the intensity of the digitalis action, and this was particularly satisfactory in that group of patients who are in this respect very sensitive to the drug, in whom marked changes in the ventricular rate occur readily when the drug is given or withheld.

Two assumptions form the basis of the present study: first, that if a given degree of depression of conduction has been produced by digitalis and this effect is not increased in intensity by the administration of a fixed daily dose for weeks or months and no toxic symptoms occur, the patient is capable of eliminating that dose; second, if a fixed daily dose of digitalis produces progressively increasing depression of conduction, that dose is greater than the patient is eliminating. Thirty-four satisfactory tests were carried out on a series of 23 patients.

The authors conclude that the daily excretion dose is a misconception and that the amount of digitalis eliminated in a day cannot be stated as a given quantity under all conditions but varies with the amount of the drug present in the body.

Parkinson, John, and Campbell, Maurice: The Quinidine Treatment of Auricular Fibrillation. Quart. J. Med. 22: 281, 1929.

The object of this study has been to assess the value of quinidine in the treatment of auricular fibrillation in hospital and private practice and to see what advantages it may possess. The authors have studied 20 patients with paroxysmal and 44 with established fibrillation in whom quinidine seemed more appropriate than digitalis. As they were selected as occasion rose during five years, it is obvious that only a small proportion were thought suitable for this treatment.

Nine out of 18 cases of paroxysmal fibrillation were much improved, the attacks being abolished or greatly reduced for as long as the patient was under observation. In one-fourth of the cases the advantage was only temporary, and in the remainder there was no improvement at all. Quinidine, therefore, is the best treatment for this type of fibrillation whenever the attacks are of such length or frequency as to justify continuous medication provided the patient is well enough in the intervals. Its use for this purpose is devoid of risk.

In selecting patients with established fibrillation as suitable for treatment with quinidine, attention was paid to the etiology, to the duration of fibrillation and to the clinical condition, especially to the size of the heart and the signs of failure. The results were best of all where there was no rheumatic valvular disease and no hyperthyroidism, for all those in whom the arrhythmia was the only evidence of heart disease still have normal rhythm after an average period of two years. In these 44 selected patients with established fibrillation, normal rhythm was restored in 30 (68 per cent). In 23 patients, more than half of all who were treated, normal rhythm not only was restored but also was still maintained after an average period of more than two years and in one patient after five years. Four of these patients had short temporary relapses which were readily brought to an end with quinidine. The other seven of the thirty successes kept well for an average period of eighteen months before they reverted to fibrillation. With two exceptions all patients were strikingly better for the return of normal rhythm. Some were much better than they had been previously when taking digitalis, and many are now living such active lives that it is difficult to think that they could have been equally well with digitalis treatment.

Severe toxic effects were seen in two patients. Embolism, which occurred in two others but left no permanent disability, was found no more frequently in quinidine than under digitalis treatment. One patient died suddenly while taking quinidine.

When fibrillation has been established for more than six months, when there is a large heart or gross congestive failure, or when there is active rheumatic or syphilitic infection, there is more danger in using quinidine and less chance of lasting success.

The onset of fibrillation in a patient enjoying good or even moderate health should be regarded as a medical emergency. Quinidine, if it is used at once, is generally a safe and effective remedy and normal rhythm may be maintained for years.